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SURGERY

A Monthly Journal Devoted to the Art and Science of Surgery

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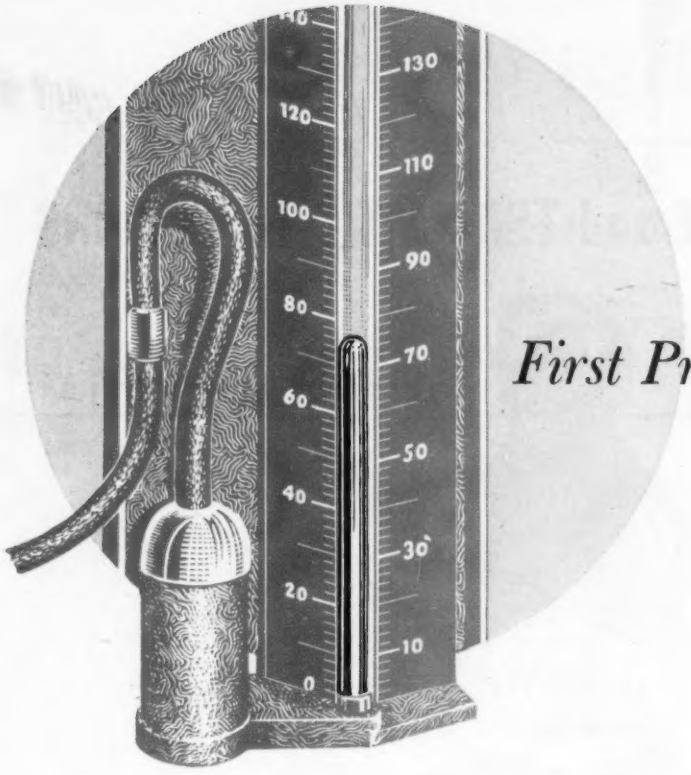

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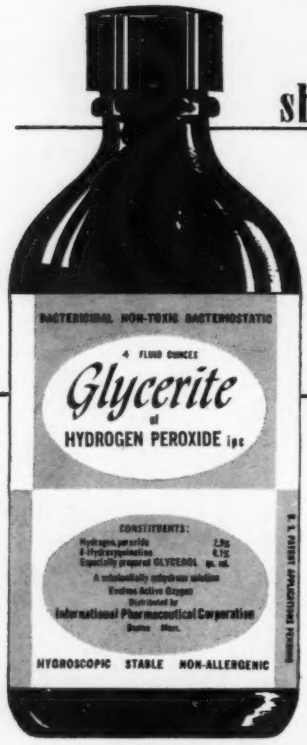

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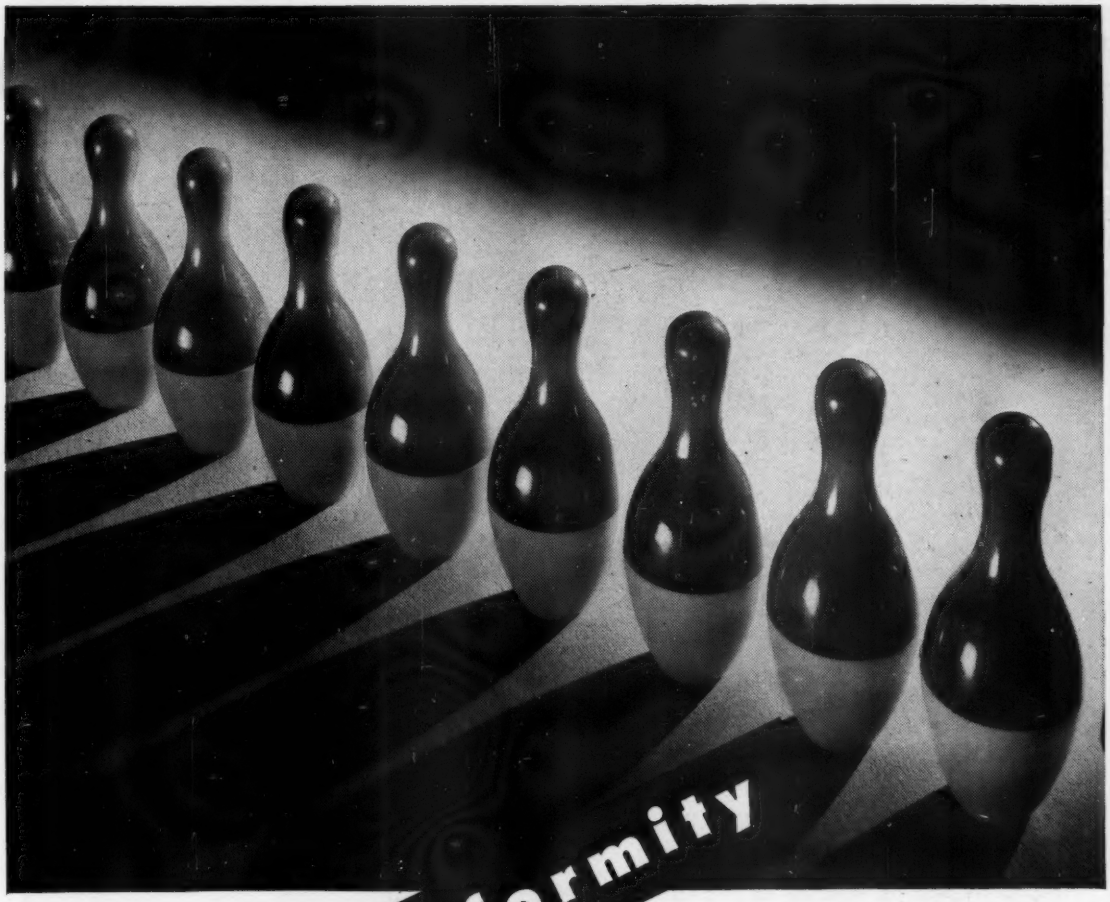

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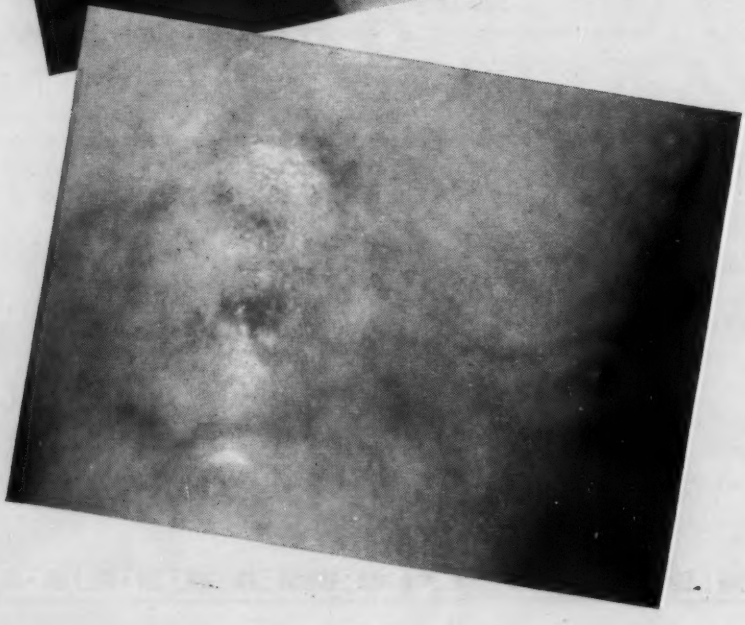

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The remainder of the book has, of course, been checked and revised. The chapter on the Intestinal Tract in Relation to Gynecologic Surgery and the chapter on Anesthesia have been rewritten by their author, Dr. H. S. Brookes.

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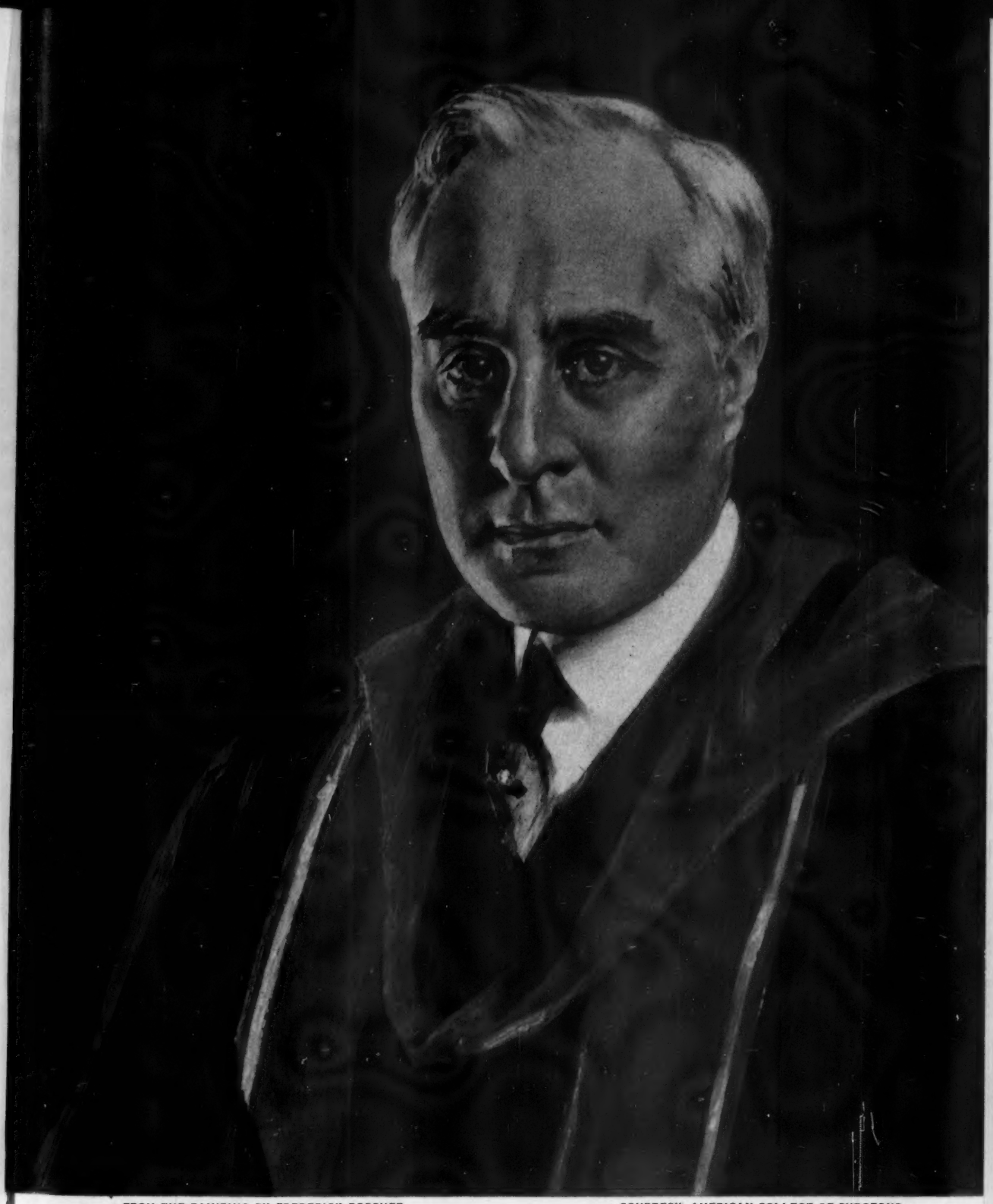

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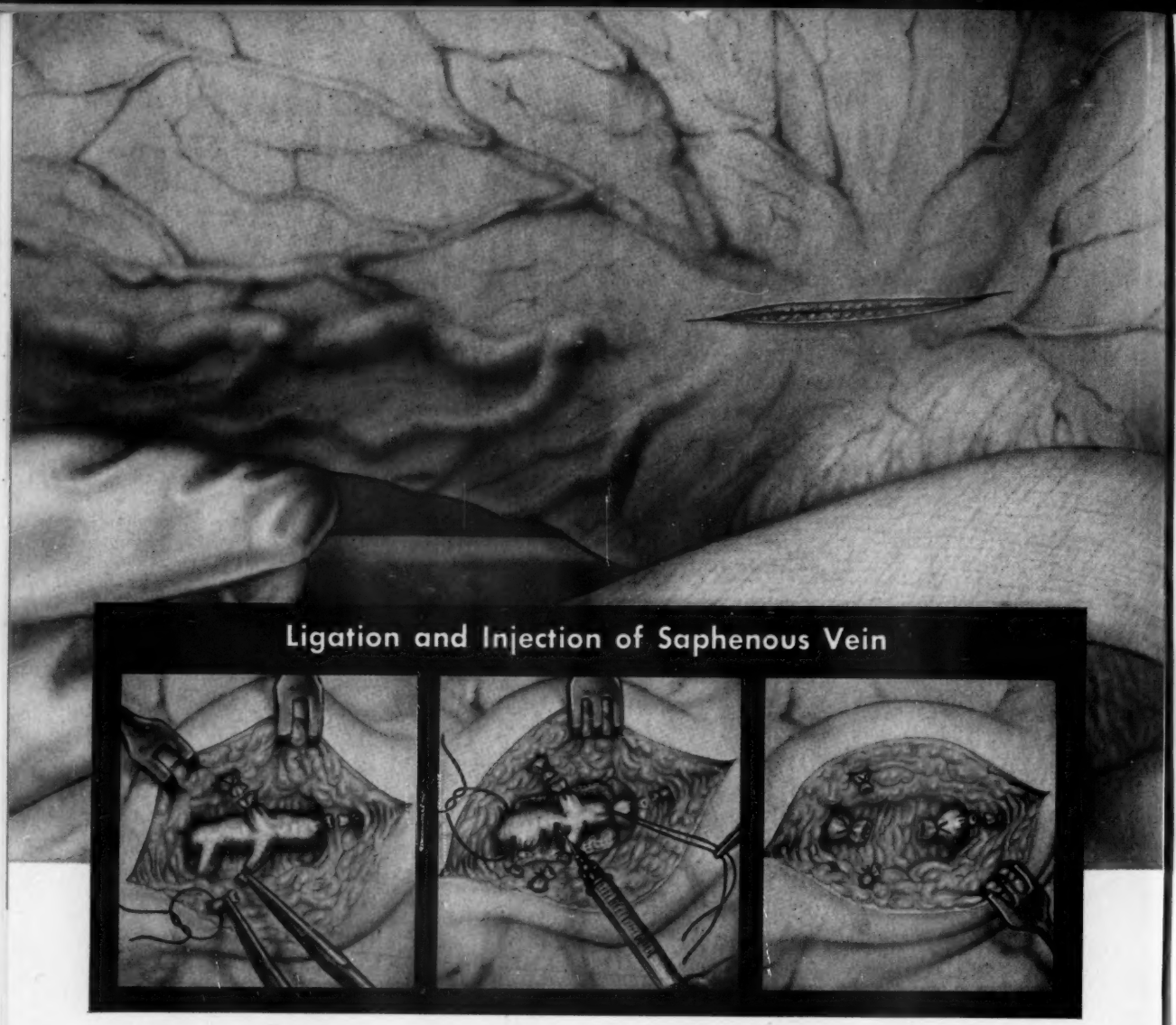

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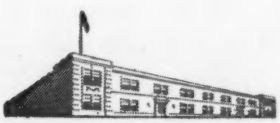

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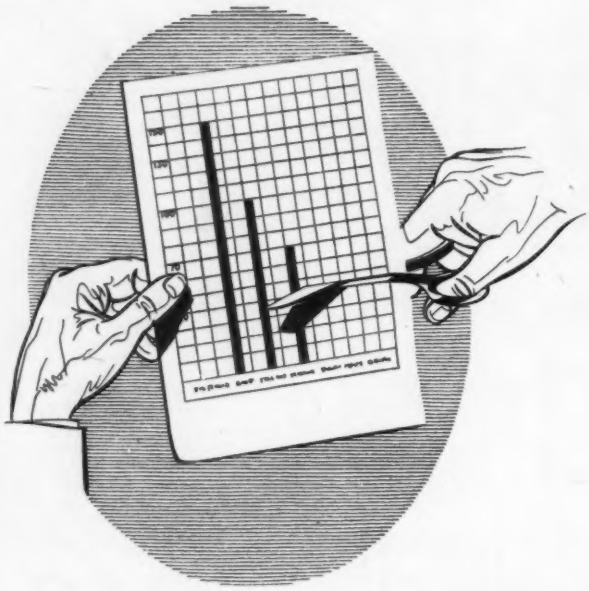

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SURGERY

VOL. 23 FEBRUARY, 1948 NO. 2

Original Communications

THE OPERATIVE TREATMENT OF CHRONIC GASTRIC AND DUODENAL ULCER

CHRISTIAN BRUUSGAARD, M.D., OSLO, NORWAY

(From the Ullevaal Hospital, Surgical Department III)

FOLLOW-UP EXAMINATIONS OF PATIENTS TREATED WITH GASTROJEJUNOSTOMY AND PARTIAL GASTRECTOMY

I COMPILED,* in 1946, the following results and drew the conclusions listed here after clinical, roentgenologic, and in part gastroscopic follow-up examinations of a total of 600 patients treated surgically for chronic gastric and duodenal ulcer at the Oslo University Clinic Department A, and Aker Hospital. The period of observation for most of the patients was over four years.

1. Gastrojejunostomy was done in 416 patients operated upon. The mortality rate was 4.7 per cent. During the time gastrojejunostomy was used as a routine method, the mortality rate was 1.4 per cent in 139 operations.

2. Partial gastrectomy was done in 572 patients operated upon. The operative method was partial resection with gastrojejunostomy according to modifications of the Billroth II method. The mortality rate was 4 per cent in 572 resections. Partial gastrectomy with exclusion of a duodenal (pyloric) ulcer according to Finsterer's method was used in 16 per cent of the cases in this group. In one-half of the cases the resection was made through the pyloric antrum without excision of the antral mucosa, and in the other half through the pylorus or the duodenum.

Gastric Acidity.—Caffeine test meal fractionally recovered every ten minutes for two hours showed the findings listed in Table II.

Postoperative Anemia.—A hypochromic anemia not caused by gastric hemorrhage occurred in approximately 50 per cent of the women with anacid gastric contents in age groups between puberty and the menopause. In women of older age groups with anacid gastric contents and in men the percentage of hypochromic anemia was 30. This anemia responded to large doses of iron in most cases. In only 5.4 per cent did the anemia produce subjective symptoms in the patients with resection.

Received for publication, May 1, 1947.

*In Acta Chir. Scandinav. 94: Suppl. 117, 1946.

TABLE I. CHIEF POSTOPERATIVE COMPLICATIONS

COMPLICATIONS	GASTROJEJUNOSTOMY		PARTIAL GASTRECTOMY	
	FREQUENCY (%)	MORTALITY (%)	FREQUENCY (%)	MORTALITY (%)
Hemorrhage	2.4	0.5	2	0.2
Peritonitis	0.5	0.5	3	2.0
Severe evacuation difficulties	2.4	1.2	2	0.35

Complications Caused by the Changed Evacuation Conditions in the Stomach (Dumping Stomach).—The characteristic symptoms of "dumping stomach" are nausea, fainting, weakness, heaviness in the legs, and occasionally sweating. The symptoms cease in the course of about twenty minutes. As a rule the patient must lie down. If he eats while reclining or lies down immediately after meals, the symptoms do not appear. This trouble is closely connected with the first half hour following meals, chiefly breakfast and dinner.

TABLE II. THE FOLLOW-UP EXAMINATIONS OF THE PATIENTS

LOCATION	GASTROJEJUNOSTOMY (PER CENT ANACID)	PARTIAL GASTRECTOMY (PER CENT ANACID)
Duodenal ulcer	20	92
Ulcer of the pyloric region	40	95
Ulcer of the corpus region	57	100

There is no connection with hypoglycemia. The treatment, eating meals in a reclining position, is simple but not easy to follow under all circumstances. This complication occurred in 15 per cent of the gastrectomy patients, but in only 2 per cent was it so severe as to cause the patient to be listed as not cured after the operative treatment.

TABLE III. RESULTS OF OPERATIVE TREATMENT

TREATMENT	NUMBER OF PATIENTS FOLLOWED	PER CENT SYMPTOM-FREE
Gastrojejunostomy	234	64
Partial gastrectomy	364	84

Conclusions.—Gastrojejunostomy should not be used as the routine method for treatment of chronic gastric and duodenal ulcers. In my studies, gastrojejunostomy in the age group over 60 years gave the same results as partial gastrectomy in the treatment of duodenal ulcer. In patients over 60 years of

TABLE IV. INCIDENCE OF THE LATE COMPLICATIONS

COMPLICATIONS	GASTROJEJUNOSTOMY (%)	PARTIAL GASTRECTOMY (%)
Postoperative jejunal ulcer	8	
Recurrent ulcer in stomach and duodenum	9	1.8
Hemorrhage	4	
Gastritis	9	4.6
Hypochromic anemia with subjective symptoms	2.5	5.4
Evacuation disturbances, intestinal disturbances, cancer in the operated stomach, intestinal obstruction	3.5	4.2

age with duodenal ulcers, whose general condition is poor or in whom technical difficulties for performing a radical gastrectomy are present, a gastrojejunostomy can be done with little risk for recurrent ulcer or stomal ulcer.

Partial gastrectomy should be the method of choice in the treatment of chronic gastric and duodenal ulcers with the limitations stipulated for the age group over 60 years. The basis for advising this operative intervention is that the mortality rate can be maintained at a reasonably low level, that is, not over 1 to 2 per cent.

FACTORS INFLUENCING THE OPERATIVE MORTALITY RATE

What can the surgeon do to lower the mortality rate? In the following paragraphs I shall give an account of the measures which we have taken at Ullevaal Hospital, Surgical Department III, in order to keep the mortality rate down. I shall report the statistics from this department for the last four years on patients with surgical ulcer. This group includes a total of 240 patients, 32 women and 208 men, operated upon between 1942 and 1945, inclusive. The sites of the ulcers as follows: 110 duodenal (54 anterior wall, 20 posterior wall, 36 kissing ulcers), 28 pyloric, 71 in the angle of the corpus, 5 in the oral half of the corpus, 13 postoperative jejunal, and 13 in both the stomach and duodenum. Fifty-five of the patients were over 50 years of age.

Preoperative Treatment.—Careful clinical, hematologic, x-ray, and functional examinations must be made before the operation is undertaken. Poor nutritional condition should be improved, if necessary, with parenteral administrations controlled by determinations of blood proteins and electrolytes. Anemia should be repaired by blood transfusions. The patient should not be operated upon during a period of intercurrent disease or of acute gastric pain while the gastritis and inflammatory process around the ulcer are most marked, but should be given a course of medical treatment, if necessary, with gastric lavage, for a few weeks before the operation.

Operative Indications.—Operation must be done in cases with perforation, grave stenosis, repeated hemorrhage, and gastric ulcers suspected of being cancer. All ulcers which give troublesome or serious symptoms in spite of expert medical treatment should be removed surgically unless there are definite contraindications. The surgical treatment should not be postponed until the ulcer becomes callous and penetrating, thus unnecessarily increasing the operative risk.

Anesthesia.—We have used spinal anesthesia with a novocain-pantocain solution of a greater specific gravity than the spinal fluid, usually 200 mg. novocain + 10 mg. pantocain. This anesthetic lasts from one and one-half to two hours. Since our operations usually last about two hours, we have used local anesthesia in the abdominal wall in order to obtain relaxation for closure. About one-half hour before we begin the closure of the abdominal wall, a solution of ½ per cent novocain and ½/1000 pantocain is injected preperitoneally and subcutaneously. This local anesthesia, which also has a general effect, has given excellent results. We have used N_2O ether anesthesia in only a few cases.

Choice of Operative Method.—Local pyloroplastic operations were used only twice during this period. The moderate mortality rate of these operations does not outweigh the more certain results obtainable by the radical operations.

Gastrojejunostomy was used in patients over 60 years of age for treatment of duodenal ulcers, when there was poor general condition or technical difficulty to add to the operative risk.

Partial (about two-thirds) gastric resection according to Billroth II has been the routine method in the treatment of gastric and duodenal ulcers. My previous investigations have shown that the different end-to-side gastrojejunostomies commonly used in the Billroth II resections all give the same satisfactory results, therefore the surgeon has free choice of method in this respect. The important point is that he chooses one method and becomes expert at it. At Ullevaal Hospital, Department III, we have used the Krönlein modification of Billroth II during this four years. After the usual resection through the upper part of the duodenum, the stomach is mobilized. The resection line is then made perpendicular to the axis of the stomach. The amount of the stomach removed in our cases has not been weighed, but the resection has been from 60 to 75 per cent of the stomach. We are of the opinion that the gastrectomy should be a 75 per cent resection to accomplish a satisfactory operation for ulcer.

The finishing gastrojejunostomy is in our cases made terminolaterally. The afferent loop is made a maximum of 20 cm. long, and placed in an antecolic position from the lesser to the greater curvature. Extensive experimental work from the University of Minnesota Hospitals by Wangensteen and his associates, has shown that a short afferent anastomotic loop is of great importance for a satisfactory result from the gastrectomy. Wangensteen in his operations uses a 2 to 5 cm. afferent loop placed in retrocolic position. I am of the opinion that our operative technique would be nearer "the satisfactory operation for ulcer" if we made our antecolic afferent loop about 10 to 12 cm. long or made a retrocolic anastomosis with an afferent loop 5 to 8 cm.

IMPORTANT TECHNICAL DETAILS IN THE PERFORMANCE OF KRÖNLEIN'S MODIFICATION OF PARTIAL GASTRIC RESECTION ACCORDING TO BILLROTH II

The freeing of the duodenum and the closure of the duodenal stump are the most important points in the accomplishment of the Billroth II resection. When there are no particular difficulties, the duodenal cuff which is to be inverted should be about 2 cm. long and should be closed with one continuous or interrupted catgut suture and two silk purse-string sutures. Callous ulcers, particularly those which penetrate into the pancreas, demand special skill and care in mobilization. In some cases it has been necessary to leave the base of the ulcer at its point of penetration into the pancreas. The mobilization of the duodenum is then continued beyond the distal edge of the ulcer, if possible, in order to obtain a cuff ½ to 1 cm. wide with which to cover the resection surface. The anterior wall of the duodenum has been used in so far as possible, and every effort has been made to avoid injury to the pancreas during the inversion of the duodenal stump. In the event that the pancreas has been injured during the procedure, or if the security of the duodenal stump closure is questionable, a

drain has been placed on the duodenal stump and allowed to remain a week. In cases in which the duodenal stump closure might be insecure, an anastomosis between the afferent and efferent loops can be used to relieve the duodenum. There has been only one case of duodenal insufficiency in over 200 radical resections. In this case a duodenal fistula formed and healed spontaneously in two weeks.

Partial gastrectomy with exclusion of a duodenal (pyloric) ulcer according to Finsterer's method has been performed in only three cases, and then as a prepyloric resection. It is important to remove the mucous membrane in the excluded distal part of the stomach. The blood supply to this part of the stomach must be left intact. The closed stump of this antral pouch may be sewed to the anterior abdominal wall. The excluded central part of the stomach must not be made less than 5 cm. long.

Our material shows that a radical removal of a duodenal or pyloric ulcer can be done in almost all cases with little risk.

The Anastomotic Suture.—Before the suture is commenced it is important that the curvatures at the place of the anastomosis are made as free as possible of omental fragments.

We use three layers of sutures in the anastomosis. In the serosa we use an interrupted silk suture and a continuous catgut suture. After the seromuscular layer is divided, all the visible blood vessels are ligated with a suture ligature, a sufficient amount of the seromuscular coat being engaged to prevent the stitches from cutting through. For the third suture the mucosal layers are separated and a continuous catgut suture is made through all layers of the stomach. This suture is made carefully with comparatively small stitches. Particular care is taken with the corner stitches in order to ensure free entrance to, and exit from, the stomach. In most cases we have used clamps on the stomach and duodenum while making the anastomosis. We have not used clamps in a number of cases and have encountered no disadvantages as a result.

My earlier investigations mentioned previously showed that the immediate operative results were the same in Billroth II operations with three and with two layers of sutures in the anastomosis. Wangensteen uses one layer of interrupted silk sutures in closed gastric anastomoses. The three layer suture method has given very good immediate results in our gastric resections, and a very small amount of emptying disturbances, but experience shows that a one-layer anastomosis as done by Wangensteen can be done with the same degree of safety. Obviously, a single-row inversion provides large patulous orifices in the new stoma.

SURGICAL TECHNIQUE IN THE TREATMENT OF POSTOPERATIVE JEJUNAL ULCER

The operative treatment of postoperative jejunal ulcer should be so radical as to produce anacidity. This is attainable only by a radical gastrectomy and resection of the previously excluded pyloric antrum in cases in which Finsterer's operation has been done. The anastomosis must be a short loop anastomosis. We have tried to avoid resection of the anastomotic loop of the jejunum by using the previous anastomosis in several cases. By these procedures we have

avoided the more dangerous resection of the jejunum with end-to-end suture of the resected surfaces.

Postoperative Care.—Atony of the stomach and intestines and swelling of the new anastomosis require careful regulation of the oral fluid intake and supplementary rectal and parenteral fluids, as well as blood infusions, when necessary, controlled by determinations of the patient's blood protein and electrolyte levels. The patient should have regular gastric aspirations morning and evening, or oftener, if necessary, for the first three days. We use a thin nasal tube, with no continuous suction. *The patients usually get up on the second or third postoperative day.*

THE IMMEDIATE POSTOPERATIVE RESULTS

Operative Complications.—Postoperative peritonitis occurred in one case as a Douglas abscess which healed after drainage. *Leakage from the duodenal stump occurred once after partial gastrectomy.* Serious postoperative hemorrhage occurred once, but the patient recovered after blood transfusions. Pulmonary embolism occurred once *with fatal outcome.* This patient did not get up until the seventh day, and he died the moment he arose. Thrombosis has been practically eliminated by allowing the patients to get up soon after the operation. One patient had a peculiar nutritional disturbance with muscular atrophy and paralysis after the operation.

Mortality Rate.—In the 240 stomach operations which include 2 pyloroplasties, 23 gastrojejunostomies, 3 resections for exclusion, and 212 partial gastrectomies, there has been one death (pulmonary embolism). This gives a mortality rate of 4 per thousand. The operations have been performed by nine surgeons using the same operative technique.

The operations and the operative techniques used in Department III of Ullevaal Hospital have given good immediate operative results with a low mortality rate, as is shown from my study. The operations include a relatively large number of gastrojejunostomies, 10 per cent (used for the treatment of duodenal ulcer on elderly persons), while resections for exclusion (Finsterer) have been used only exceptionally. The partial gastrectomies have been radical, about 75 per cent resections in most cases, but with a proximal anastomotic loop up to 20 cm. in length.

In spite of the satisfactory immediate results obtained there are two criticisms of the operative methods used which can be made: Gastrojejunostomy has been performed in too many cases, and the proximal anastomotic loop in the partial gastrectomies has been too long.

The end results cannot be discussed because the patients were operated upon so recently that there have been no adequate follow-up examinations.

SUMMARY

I have reported the immediate and late results in a study of gastric and duodenal ulcers treated operatively. I have discussed measures employed to achieve a lower operative mortality rate.

PRODUCTION OF ALLERGIC GASTRIC AND DUODENAL EDEMA WHICH PREDISPOSES TO THE HISTAMINE-PROVOKED ULCER IN DOGS

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GASTROINTESTINAL manifestations of allergic disease were recognized by Osler[1] in 1904, when he described visceral crises in allergic individuals to be due to infiltration of blood and serum into the wall of the gastrointestinal tract. In 1915, Crispin[2] observed by x-ray, in a patient with hematemesis, a transient lesion at the pylorus which was found at operation to be due to angioneurotic edema. Roentgenologic evidence of the disturbances which occur in allergic reactions of the gastrointestinal tract has been obtained by others,[3, 4] gastric retention being the most common finding. Hyperemia, edema, and submucosal hemorrhage in the stomach have been observed gastroscopically in experimentally reproduced gastric allergy in man.[5]

Lintz[6] observed that patients with allergy frequently show evidence of gastric or intestinal hemorrhages. Of forty-four patients with bronchial asthma examined by him, eighteen had blood in the stomach contents; ten of twenty-five, in the feces. Of 300 patients with general allergy, 95 also had gastrointestinal allergy. In thirty-two unselected patients with peptic ulcer, Kern and Stewart[7] found that 40 per cent of those patients gave a history of some type of allergic disturbance. Gay[8] was able to determine, in a small series of ulcer patients, the allergens (chiefly wheat, eggs, beef, and milk) which precipitated the ulcer symptoms in these patients, by studying the leucopenia, fall in gastric acidity, and subjective discomfort of the patients. Upon elimination of the allergens from the diet, clinical improvement in these patients was noted.

Gastrointestinal changes in experimental anaphylaxis in animals were first noted by Schittenhelm and Weichardt[9] in 1910. They observed numerous miliary hemorrhages in the mucosa and submucosa of the gastrointestinal tract in sensitized dogs reinjected with egg protein. Experimental production of gastric ulcer by local anaphylaxis utilizing the principle of the Arthus phenomenon has been described by Shapiro and Ivy.[10] These investigators injected antigen into the gastric mucosa of previously sensitized dogs and rabbits; ulcers were produced which later tended to heal.

Freeman,[11] in 1925, was the first to demonstrate that local passive sensitization of the tissue followed by intravenous injection of the sensitizing antigen

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results in edema of that tissue. He injected into the skin of a normal patient serum obtained from a patient with hay fever. After two weeks the sensitizing pollen was injected intravenously, producing edema at the local site of sensitization. This phenomenon has been demonstrated experimentally in monkeys and in man by others.[12] It has been shown that serum (obtained from an allergic patient), when injected locally into the gall bladder wall, skin, ileum, rectum, and stomach, and later followed by systemic administration of the sensitizing antigen, results in pallor, hyperemia, and transient edema.

Edema of the gastrointestinal tract is often seen in conjunction with, or as a result of, other conditions which have been found to predispose to erosion and ulceration. These include the nutritional edema of hypoproteinemia,[13] portal hypertension,[14] and systemic disease such as toxemia and septicemia.

The purpose of this study, therefore, is threefold: (1) to determine whether local antigen-antibody reactions can be utilized to produce prolonged gastric and duodenal edema, (2) to determine whether edema per se (without alteration of the general condition of the animal) favors the experimental production of the histamine-in-beeswax provoked ulcer, and (3) to determine whether local anaphylaxis produced by passive local sensitization of the gastric and/or duodenal mucosa with local antigen-antibody reaction abets the ulcer diathesis. It has been found by Hay and associates[15] that forty days are necessary to produce ulcers consistently and with regularity in dogs given histamine-in-beeswax daily. In their series, the average appearance time of the ulcers after the administration of histamine was begun was twenty-three days.

METHOD AND MATERIALS

Serum was obtained from an adult horse which had been sensitized previously to five different antigens, these being swine serum (anticholera hog serum), bovine serum (antihemorrhagic septicemia serum of bovine origin), rabbit serum, egg albumin (1:10 dilution), and skim milk. The horse was given intravenous injections of each antigen every other day for three weeks in 40 to 50 c.c. doses. Thirty-four days after the first injection, when intradermal wheals indicated marked sensitivity of the animal to all antigens, blood was drawn for serum extraction and preserved with aqueous merthiolate in 1:10,000 dilution. Antibody titers (precipitin method) of the horse serum for the different antigens were as follows:

Swine serum	1:6400
Bovine serum	1:3200
Rabbit serum	1:1600
Egg Albumin	1:6400
Skim milk*	------

Adult dogs were subjected to laparotomy under sodium pentobarbital anesthesia, and, with the exercising of sterile precautions, the horse serum was applied locally to the stomach and/or duodenum, either by direct infiltration into the submucosa (20 c.c.), or by intra-arterial (left gastric artery) injection (40 c.c.), the horse serum in the latter instance reaching the gastric wall via the blood vessels of the stomach. The area of infiltration was marked with a loose

* It was not possible to determine the antibody titer to skim milk by the precipitin method employed.

cotton stitch just into the mucosa. The size of the area sensitized was about 25 sq. cm. when direct infiltration was used, and approximately two-thirds of the stomach when intra-arterial injection was employed. After one to ten days, laparotomy was again performed and the stomach and/or duodenum opened at a site away from the previously sensitized area for observation. One of the antigens to which the dog had been sensitized was injected systemically via a tongue vein, and measurements by a caliper of the viscus wall thickness and biopsies were obtained before and after injection of the antigen up to ten hours. The abdomens of the animals were closed aseptically and the dogs were allowed to recover; some were sacrificed in twenty-four hours for measurements and biopsies of the stomach and/or duodenum. In all experiments surgical trauma was directed away from the sensitized sites (opposite gastric wall, etc.).

This method of production of the allergic phenomenon differs from that described by Shapiro and Ivy,[10] in that in this study the animal is sensitized passively and locally (stomach and duodenum) by local injection of the antibodies, followed later by systemic administration of the antigens, instead of the active sensitization of the animal followed by local injection of the antigen according to the Arthus phenomenon.

To evaluate the role of edema thus produced upon ulcer provocation, a number of dogs were sensitized in the manner described. Forty-eight hours following sensitization, daily intravenous injection of the antigen into the systemic circulation was begun; that is, a different one of the five antigens to which the horse had been sensitized was injected daily until all five foreign proteins had been administered (a total of five days). Histamine-in-beeswax mixture, prepared after the method of Code and Varco,[16] was injected intramuscularly (30 mg. base) each evening. The only restriction of food intake exercised was elimination of milk and horse meat from the diet. One series of dogs was subjected to the same procedures, using instead of the prepared horse serum with high antibody titer, normal horse serum having a negative antibody titer to the antigens employed. Animals were sacrificed after five daily injections of antigen, and sections of the stomach and duodenum were obtained for microscopic study.

EXPERIMENTS

Experimental studies were divided into two parts as follows:

I. *Production of a Prolonged Gastric and Duodenal Edema.*—Eight dogs received a local injection of 20 c.c. of horse serum with high antibody titer directly into the gastric and duodenal mucosa and submucosa. In two dogs, the duodenum was sensitized by placing 20 c.c. of horse serum within the lumen of the duodenum, an area of approximately 5 cm. being isolated by rubber-shod clamps for twenty minutes and then released. The local reaction to systemically injected antigen (20 c.c.) in these dogs was studied in fourteen experiments from one to ten days following sensitization.

Five dogs received a local injection of 20 c.c. of horse serum with negative antibody titer directly into the gastric and duodenal mucosa and submucosa, and observations were made after systemic injection of 20 c.c. antigen in one to three days following sensitization in nine experiments.

Six dogs received local intra-arterial (left gastric artery) injection of 40 c.c. of horse serum with high antibody titer. Observations, after systemic injection of antigen, were made in two to six days following sensitization in six experiments.

Two dogs received local intra-arterial (left gastric artery) injection of 40 c.c. of horse serum with negative antibody titer and were studied in forty-eight hours, after systemic injection of 20 c.c. antigen.

Two dogs underwent laparotomy in which the stomach and duodenum were opened for observations and measurements during ten hours of exposure of the mucous membrane to the air. One of these dogs received systemic injection of antigen during the time of observation.

II. *Evaluation of Role of Allergic Edema on Ulcer Production.*—Three series of experiments were carried out.

Series 1: Twelve dogs were sensitized locally with immune horse serum of high antibody titer and daily intravenous administration of antigens was begun after forty-eight hours, together with daily administration of histamine-in-beeswax mixture.

Series 2: Six dogs were used in this series. The stomach of each dog was sensitized locally with horse serum of high antibody titer, followed in forty-eight hours with daily intravenous administration of antigens. No histamine was given in this series.

Series 3: Four dogs were used in this series. Normal horse serum with negative antibody titer to the antigens employed was injected locally into the submucosa of the stomach and duodenum of each dog, followed in forty-eight hours by daily intravenous administration of the five antigens, together with daily injections of histamine-in-beeswax mixture.

RESULTS

I. *Production of a Prolonged Gastric and Duodenal Edema.*—All dogs sensitized locally by immune horse serum of high antibody titer, either by direct mucosal and submucosal infiltration or intra-arterial injection demonstrated gross and microscopic edema following systemic intravenous injection of antigens. The two dogs in which sensitization of the duodenum was accomplished by injecting immune horse serum within the duodenal lumen also developed mucosal edema after systemic injection of antigen. All five antigens were found to be effective in initiating the response, and responses were obtained from twenty-four hours to ten days following sensitization. The onset of the edema varied between fifteen minutes and one hour, and persisted for as long as twenty-four hours; the maximum effect was noted at from two to six hours. The edema represented an increase in thickness of the viscus wall from two to five times its original thickness.

The animals which received local injection (direct infiltration or intra-arterial) of horse serum with negative antibody titer and were observed after intravenous administration of antigens demonstrated no edematous reaction, and the findings were uniformly negative.

Exposure to the air of the gastric or duodenal wall for ten hours, with or without intravenous injection of antigen, in dogs not passively sensitized resulted in no more than 1 mm. increase in thickness.

Microscopic examination revealed marked "watery" edema located chiefly in the submucosa. There was no demonstrable cellular reaction and eosinophiles were not found to be increased in number.

Fig. 1.—Microscopic section demonstrating submucosal gastric edema in a dog subjected to local passive sensitization and systemic injection of antigen (magnification ×7).

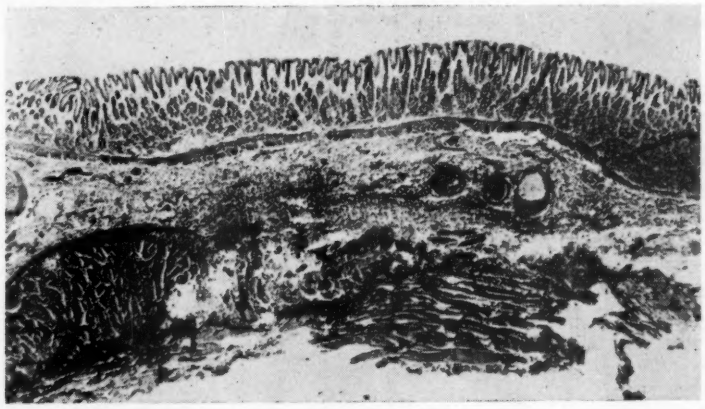

Fig. 2.—Microscopic section of normal gastric wall of a dog (magnification ×7).

II. *Evaluation of Role of Allergic Edema on Ulcer Production.*—

Series 1 (see Table I): All of the twelve dogs, subjected to the production of local anaphylaxis with concomitant administration of histamine-in-beeswax mixture, demonstrated edema with ulcer and/or erosion at the site of sensitization at the time of death or sacrifice (five days or less). Two dogs died of peritonitis after four and five days of antigen and histamine administration; one showed numerous deep duodenal ulcers in the area of edema with perforation; and the other, a perforated gastric ulcer in the area of edema.

TABLE I. DOGS SUBJECTED TO PRODUCTION OF LOCAL ANAPHYLAXIS WITH CONCOMITANT ADMINISTRATION OF HISTAMINE-IN-BEESWAX MIXTURE*

DOG	WEIGHT (POUNDS)	SENSITIZATION	NUMBER OF ANTIGENS AND INJECTIONS	NUMBER OF HISTAMINE INJECTIONS	RESULTS	REMARKS
703	29	50 c.c. left gastric artery	4	4	Gastric edema; three bleeding gastric ulcers in edematous area	Sacrificed; melena
706	35	40 c.c. submucosal infiltration: gastric and duodenal	5	5	Punctate hemorrhagic areas at edema	Sacrificed
708	26	40 c.c. left gastric artery and duodenal lumen	2	2	Three lesser curvature ulcers in edematous area	Emesis; died of aspiration pneumonia
755	32	20 c.c. submucosal infiltration: gastric and duodenal	5	5	Gastric bleeding points; duodenal ulcer; edema	Sacrificed
759	32	Same as Dog 755	5	5	Gastric erosion and ulcers in edematous area	Sacrificed
760	34	Same as Dog 755	5	5	Two small gastric ulcers in edematous area	Sacrificed
763	34	Same as Dog 755	4	4	Gastric bleeding points; perforated duodenal ulcer and other deep duodenal ulcers in edematous areas	Died, peritonitis
764	34	Same as Dog 755	5	5	Gastric ulcer in edematous area	Sacrificed
766	24	Same as Dog 755	2	2	Small gastric ulcers in edematous area	Died, pneumonia
767	29	Same as Dog 755	2	2	Gastric bleeding points and erosions in edematous areas; duodenal erosion in edematous area	Sacrificed
768	33	Same as Dog 755	5	5	Large perforated gastric ulcer in area of edema	Died, peritonitis
769	31	Same as Dog 755	5	5	Gastric ulcer in area of edema	Sacrificed

*The procedure in this series of dogs consisted of: (1) Passive local sensitization of the stomach and/or duodenum with immune horse serum of high antibody titer. (2) After forty-eight hours, daily intravenous injections of the antigens were given, accompanied by, (3) daily intramuscular injections of histamine-in-beeswax (30 mg. base).

All twelve dogs developed edema with ulcer and/or erosion in five days or less. Two dogs died of peritonitis due to perforated ulcers, one gastric and one duodenal. There was evidence of hemorrhage in five animals.

TABLE II. DOGS SUBJECTED TO PRODUCTION OF LOCAL ANAPHYLAXIS WITHOUT THE ADMINISTRATION OF HISTAMINE-IN-BEESWAX MIXTURE

DOG	WEIGHT (POUNDS)	SENSITIZATION BY SUBMUCOSAL GASTRIC INFILTRATION	NUMBER OF ANTIGENS AND INJECTIONS	NUMBER OF HISTAMINE INJECTIONS	RESULTS	REMARKS
787	29	20 c.c.	5	0	Bleeding gastric ulcer in edematous area	Sacrificed, melena
786	26	20 c.c.	5	0	Edema only	Sacrificed
796	32	20 c.c.	5	0	Edema only	Sacrificed
797	31	20 c.c.	5	0	Gastric ulcer in edematous area	Sacrificed
806	36	20 c.c.	5	0	Edema only	Sacrificed
807	30	20 c.c.	5	0	Gastric ulcer in edematous area	Sacrificed

Summary: All six dogs developed edema, three of which presented gastric ulcers, one bleeding.

TABLE III. DOGS SUBJECTED TO PRODUCTION OF LOCAL ANAPHYLAXIS WITH SENSITIZING HORSE SERUM OF NEGATIVE ANTIBODY TITER

NUMBER OF DOGS	SENSITIZATION	NUMBER OF ANTIGENS AND INJECTIONS	NUMBER OF HISTAMINE INJECTIONS	RESULTS	REMARKS
4	Stomach and duodenum	5	5	No edema, erosion, or ulcer	Sacrificed

TABLE IV. DOGS RECEIVING DAILY INJECTIONS OF HISTAMINE-IN-BEESWAX MIXTURE ALONE

NUMBER OF DOGS	SENSITIZATION	NUMBER OF ANTIGENS AND INJECTIONS	NUMBER OF HISTAMINE INJECTIONS	RESULTS	REMARKS
4	0	0	5	No erosion or ulcer	Sacrificed

Series 2 (see Table II): Three of the six dogs subjected to the production of local anaphylaxis without administration of histamine-in-beeswax mixture demonstrated definitive ulcers at the site of edema, when sacrificed after five days of antigen administration. All six showed edema at the site of sensitization. One ulcer was actively bleeding.

Fig. 3.—Duodenal ulcers (one perforated) in area of duodenal edema in Dog 763 (see Table I) subjected to the production of local allergic edema accompanied by four daily injections of histamine-in-beeswax mixture (magnification ×1.2).

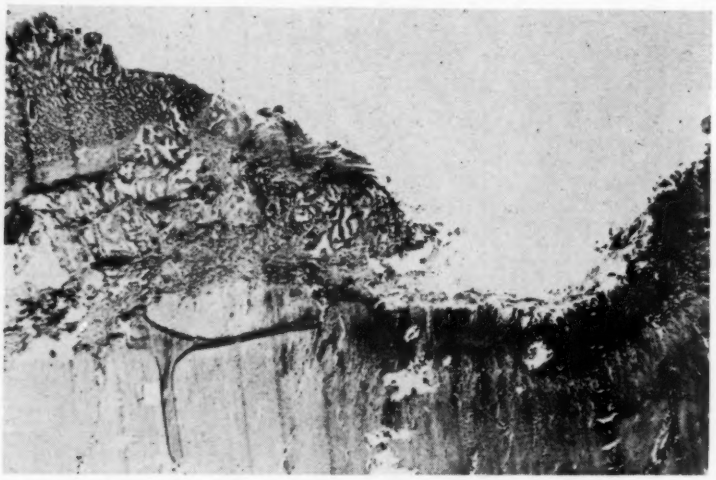

Fig. 4.—Microscopic section through edge of duodenal ulcer situated in an area of duodenal allergic edema in Dog 763 (see Fig. 3) (magnification ×7).

Series 3 (see Table III): Of the four dogs subjected to local injection of normal horse serum of negative antibody titer followed in forty-eight hours by daily systemic injection of antigen accompanied by histamine-in-beeswax administration, none showed edema, erosion, or ulcer.

Four additional control dogs (Table IV) receiving daily injections of histamine-in-beeswax mixture alone, did not present ulceration when sacrificed in five days.

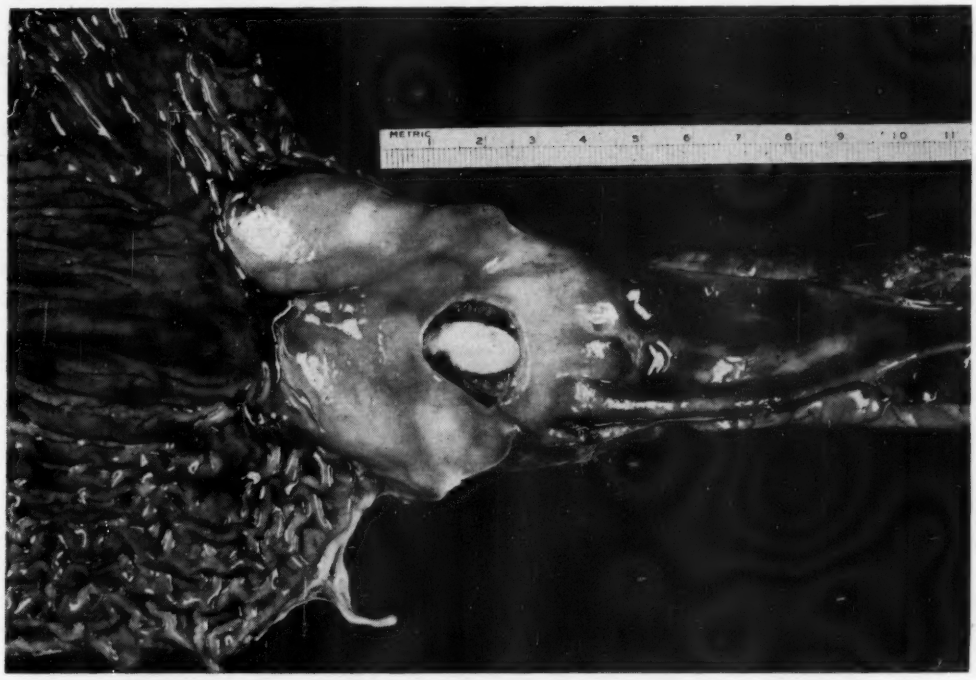

Fig. 5.—Perforated gastric ulcer in area of antral edema in Dog 768 (see Table I) subjected to the production of local allergic edema accompanied by five daily injections of histamine-in-beeswax mixture (magnification ×0.85).

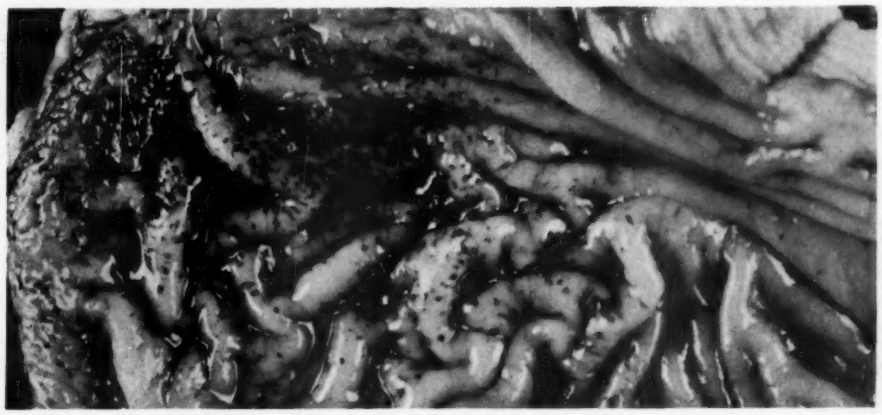

Fig. 6.—Punctate gastric bleeding points in area of gastric edema in Dog 706 (see Table I) subjected to the production of local allergic edema accompanied by five daily injections of histamine-in-beeswax mixture (magnification ×1.3).

DISCUSSION

It is seen that local antigen-antibody reaction produced by local passive sensitization and initiated by systemic administration of antigen results in marked edema of the gastric and duodenal wall. That it is a specific antigen-

antibody reaction is evidenced by the circumstance that no edematous reaction takes place when the sensitizing horse serum has a negative antibody titer to the antigens in use. When the passively sensitizing serum contains antibodies to five antigens, a different antigen may be injected daily for continual provocation of the edematous response. By this method, edema on an allergic basis can be made to persist for as long as five days when five antigens are employed for its production.

By passively sensitizing an organ and then producing an allergic edema by the subsequent intravenous injection of the antigen no general manifestations of anaphylactic shock were produced. By avoiding the appearance of anaphylactic shock, the alterations caused by local edema in the stomach and duodenum could be studied without the marked alterations of the general physiology of the body accompanying anaphylactic shock.

Fig. 7.—Bleeding gastric ulcer in area of gastric edema in Dog 787 (see Table II) subjected to local passive sensitization of the stomach followed by daily systemic injections of the antigens; no histamine administration; sacrificed in five days (magnification ×0.7).

It was noted in this study that swine serum antigen initiated the most marked response, and rabbit serum antigen, the least. This fact reflects the degree of sensitization of the horse used for the source of the sensitizing serum. It was observed that an antigen is unable to provoke a second response if reinjected into the same animal within a period of ten days.

It is apparent that ulcers are provoked more readily and quickly with histamine in the presence of gastric and duodenal edema resulting from experimentally produced local antigen-antibody reaction. It is noteworthy that ulcers were produced by local anaphylaxis alone and that ulcers were produced more consistently by administration of histamine in addition to the production of local anaphylaxis. No ulcers were produced by five daily injections of histamine-in-beeswax alone. From these observations it would appear that local anaphylactic edema can be an important factor in the production of ulcer.

The size of the sensitized or edematous area had no apparent relation to the incidence of ulceration; the presence or absence of an edematous area, on the other hand, had a distinct bearing upon the occurrence of ulceration. All ulcers were situated in the edematous areas.

The incidence of ulceration in the dogs subjected to local anaphylaxis without histamine administration was three of six. It is possible that this incidence

would be greater if serum containing antibodies for a greater number of antigens were employed, thus increasing the number of daily injections of antigens and prolonging the duration of edema.

CONCLUSIONS

1. A method of experimentally producing a prolonged gastric and duodenal edema on an allergic basis in dogs is described. Edema resulting from local antigen-antibody reaction is produced by passive local sensitization to multiple antigens, and initiated by daily systemic administration of the different antigens.

2. Experimental gastrointestinal edema resulting from local antigen-antibody reaction favors the development of the histamine-provoked ulcer in dogs and abets the ulcer diathesis.

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ASEPTIC GASTRIC RESECTION, BILLROTH I TECHNIQUE

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IN THE field of gastric surgery beset with doubt and slowly paced, treated with fear and sometimes with audacity, we have advanced from Torell's partial extirpation to the total resection of Graham. With conscious boldness, based increasingly more on physiopathology and clinical experience, the surgeon sought to cure malignant tumors by extirpation of the involved part of the organ. Restoration of the anatomic relations of the organ by gastroduodenostomy was the object of former surgeons who lacked the basis for further advances in gastric resection. It occurred to them as logical, when forced to resect a segment of the organ, to re-establish continuity of the lumen by preserving the same relation and form as existed before surgery was undertaken. Thus, the dominance of anatomy over physiology and pathology was clearly shown. In this manner Kocher, Pean, and Billroth proceeded in their major preoccupation of curing patients with gastric neoplasm.

Obliged to accomplish extensive resections, however, they witnessed successive failures of gastroduodenostomy because of the difficulty of uniting the cut edges, and because of the failure of union following suture of the stomach to the duodenum under tension. Billroth overcame these difficulties through gastrojejunostomy. The two techniques of Billroth are the patterns from which the other methods of gastric resection with which we are acquainted are planned.

Lately surgeons have been divided in their choice between the first and second procedures of Billroth, but it is best to accept both methods. If the first, which is an ideal procedure, is not always applicable and thus at times fails in its results, the second because it is efficient and easy of execution does not deserve such an exclusive and general indication.

In the case of ulcers and tumors the extensive resection necessary accounts for the performance of a gastrojejunostomy. We find, nevertheless, a perfect indication for gastroduodenostomy after conservative partial resections or those that offer the possibility of reconstructing the gastroduodenal lumen under favorable circumstances. The degree of hyperacidity, gastroscopic findings, etc., is useful in the determination of the extent of resection. Thus we can by semeiotic means and by exploration during the operative procedure often plan on re-establishing the continuity once the resection is performed following the Billroth I technique.

Considering the indications for this technique instead of forgetting them with those of the past we have methodized its steps under the same principles of the aseptic gastrectomy, Billroth II technique described by Monteiro in 1945.

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METHOD

Following the same technique as the one used for mesial section in gastric resection, Billroth II method, we free the greater curvature, placing ligatures above the gastroepiploic artery. From the site for the gastric section to the duodenum, the lesser peritoneal cavity is exposed. The assistant passes two

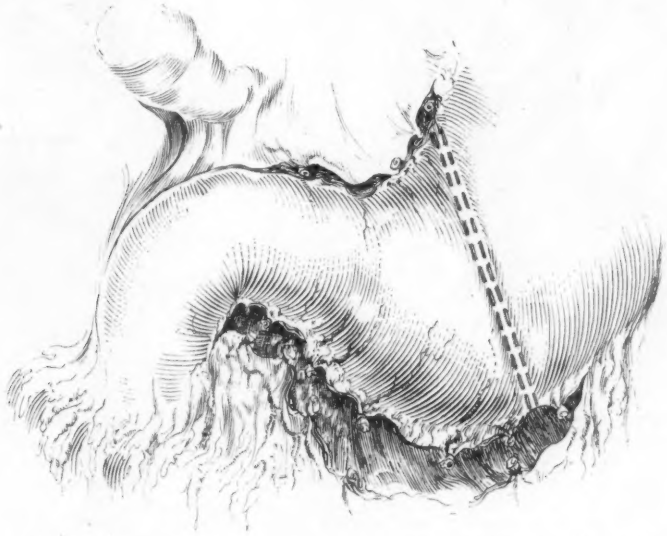


Fig. 1.—After mesial section the site for gastric section is clamped with a Von Petz instrument which leaves two rows of clips.

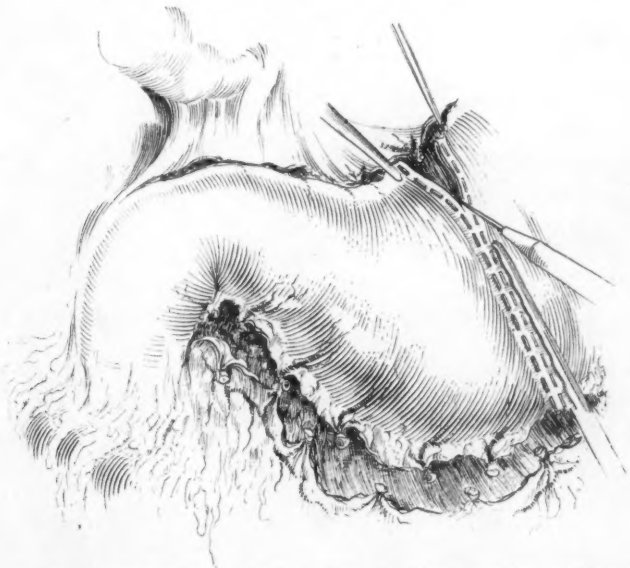


Fig. 2.—Close to the proximal margin clips is placed the anastomotic clamp. Two repair stitches are placed at the extremities and the stomach is sectioned, between clips, with the cautery.

fingers through this cavity to ascertain the pyloric vessels at the level of the lesser curvature (Finochetti). Once the vessels are tied we start freeing the portion to resect with care in hemostasis. At this moment the duodenal dissec-

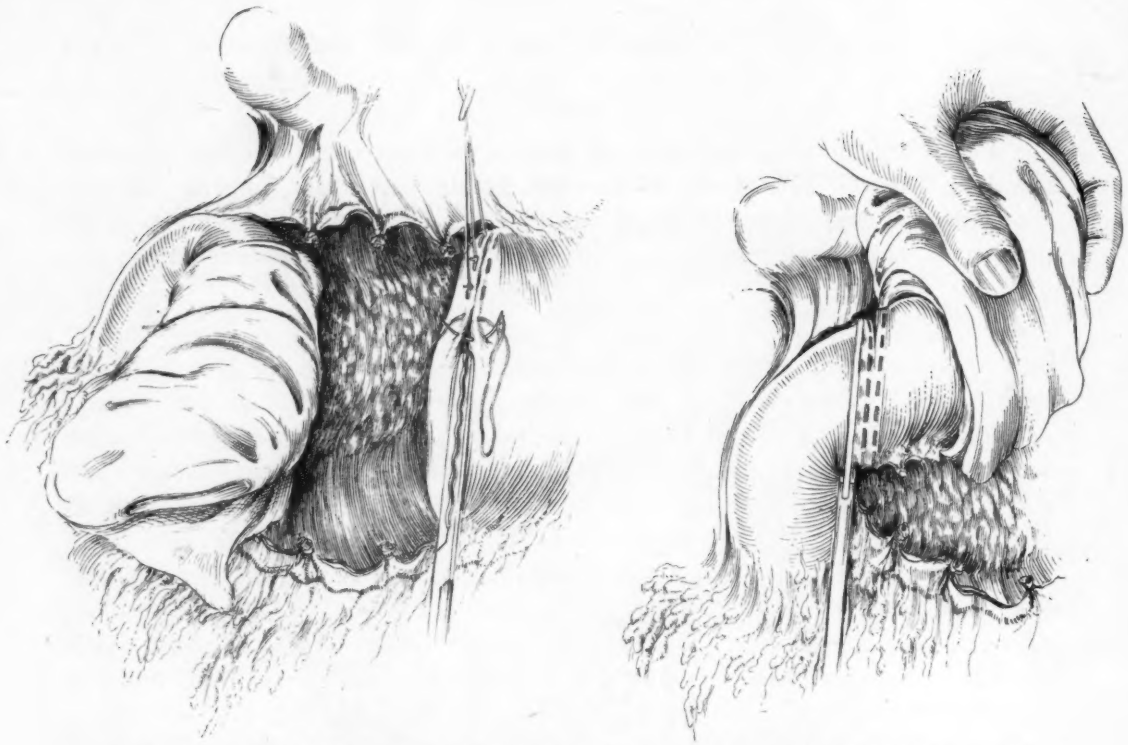


Fig. 3.—Once the distal margin is covered with gauze and riveted, the proximal margin of the stomach is inverted, which exceeds the anastomotic clamp, with Halsted stitches.

Fig. 4.—Clamping with Eugênio de Sousa's instrument, the site of the duodenum for oblique section, sectioning is carried out between clips with the cautery, after the anastomotic clamp is placed close to the distal margin.

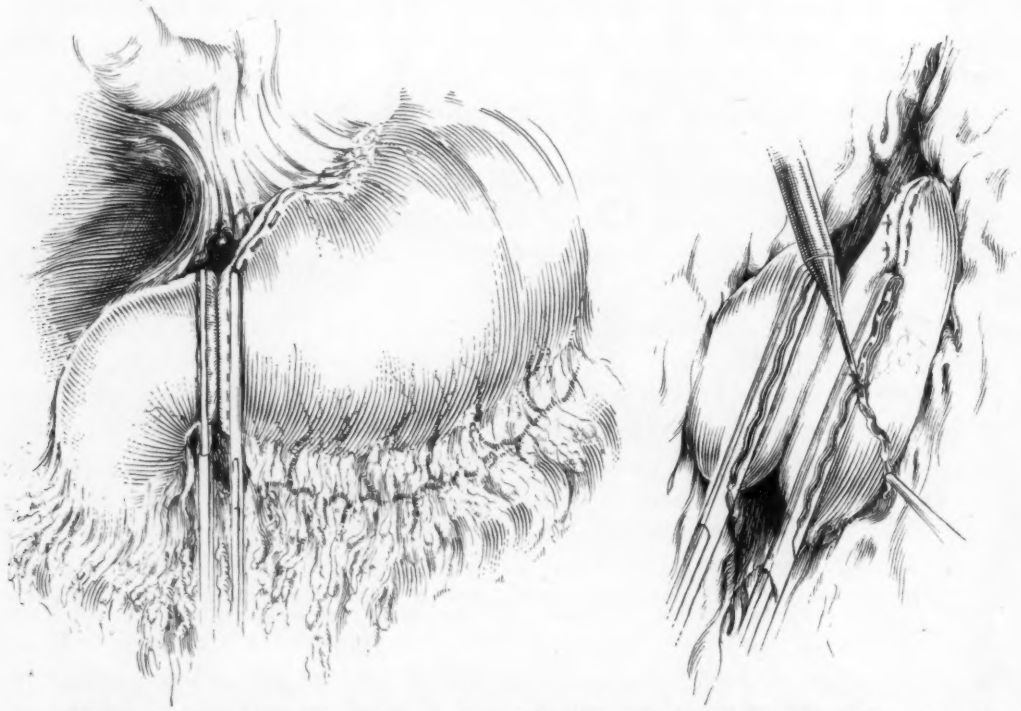
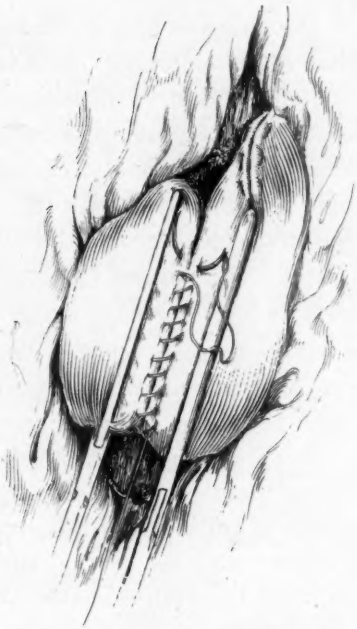


Fig. 5.—The anastomotic clamps are brought together to consider the possibility of a perfect union. Otherwise the Billroth II method can be applied.

Fig. 6.—Removal of the row of clips close to the clamp, followed by electrocoagulation of the surfaces.



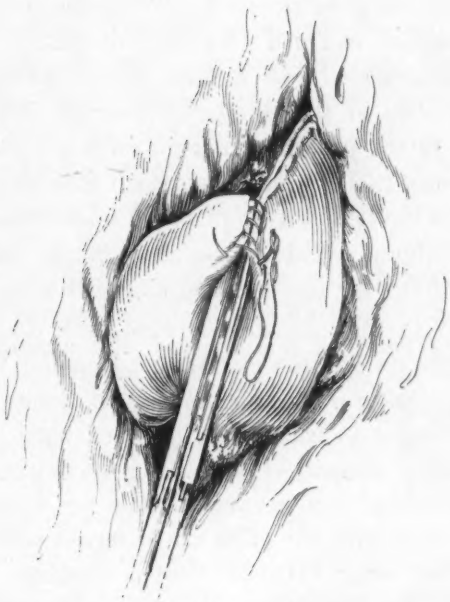


Fig. 7.—Rotation of the clamp outward, allowing the posterior seromuscular suture.

Fig. 8.—Having once outlined the superior angle, the continuous suture is followed through the anterior wall. The clamps are rotated bringing together the sectioned surfaces.

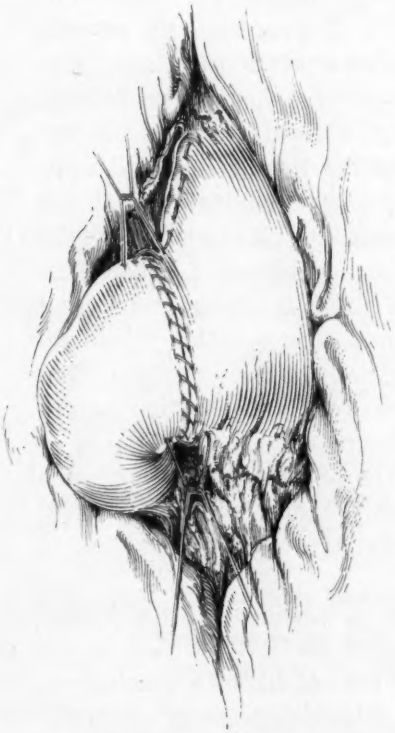


Fig. 9.—The anastomosis accomplished, reinforcement stitches are placed at the angles, especially the superior one.

Fig. 10.—View of the accomplished operation. Note the epiploplasty at the angles of anastomosis.

tion can be accomplished. When the portion of the stomach and duodenum to be resected is freed from the omentum we proceed in clamping the site for gastric section with a Von petz instrument. Repair sutures placed at the extremities of the clamped zone maintain it tense after the clamp is removed. Close to the proximal margin clips we place Monteiro's anastomotic clamp and following this we cut the stomach with the cautery. Once riveted with the distal margin covered with gauze, we invert the sectioned surface beyond the anastomotic clamp. This inversion is made with Halsted type sutures. After protecting the suture and the clamped zone we pass to the duodenal stage.

With the stomach riveted, the duodenal stage becomes easy. We use Eugênio de Sousa's clamp based on the Von Petz instrument, which facilitates the procedure. The clamping is done obliquely to afford a larger zone of anastomosis to coincide with the length of the clamp which is placed close to the distal margin clips. After sectioning between the clips with the cautery the duodenal and gastric clamps are approximated to initiate the anastomosis. The edges with the clips are removed with the cautery and the excised surfaces are electrocoagulated to effect hemostasis. By rotation the approximated clamps present the posterior walls of the stomach and duodenum for the posterior seroserous suture. This suture, which is done with nonabsorable thread, is started at the end of the clamp and carried through to the superior angle (lesser curvature). Without interrupting the continous suture, the clamps are rotated 180 degrees, bringing together the sectioned surfaces. We then continue the sutures through the anterior wall. We interrupt the suture after the angle and follow with another thread because of the possibility of a hemostatic failure. At the inferior angle (greater curvature) interrupted sutures are placed at the bases of the clamps. When the clamps are removed the thread is pulled taut, closing the opening. The suture line once accomplished is reinforced at the angles, especially the superior one (fatal suture angle). A segment of the omentum is tacked over the line of closure and the hepatogastric ligament is closed transversely. The lesser omentum is thus fixed to the stomach and duodenum to maintain both organs in alignment and secure better reinforcement at the superior angle. Figs. 1 to 10 show the details of the technique. The Von Petz instrument may be substituted by a temporary transfixation suture at the crushed zone held laterally by two strong clamps only at the portion of the stomach which is beyond the anastomosis. In the other technical steps the compression clamp and forceps effect a satisfactory closure of the sectioned surfaces meeting adequately the aseptic requirements.

DISCUSSION

The method proposed by us allows the surgeon a tactical decision in the operative procedure with choice of the first or second plan of Billroth's technique. Following gastric resection it is easy to perform a gastrojejunostomy when the conditon of the duodenum does not permit a gastroduodenostomy. Therefore, until the resection is accomplished one can plan one or the other procedure and especially consider the possibility of performing the Billroth I.

Hemostasis is of the utmost importance. Through experience in our clinic we have concluded that thorough hemostasis may very well account for the success obtained by the method of Monteiro. As is well known, the vessels in the stomach lie in the submucous layer. A perfect crushing will tear this layer together with the mucosa and circular fibers of the muscularis. When crushing does not accomplish hemostasis, the vessels which are withdrawn with the submucosa will continue to bleed into the cavity. Even if hemostasis is accomplished by crushing it may not be sufficient to withstand postoperative distention. This would result in secondary hemorrhages. It is logical to believe that the success of hemostasis and antisepsis in our method is based on the following fact: the Von Petz instrument performs the crushing but not a lamination that would pierce and permit the submucosa to retract. When the clips are withdrawn and the sectioned surface is electrocoagulated, hemostasis is affected by directly reaching the layers with the vessels. The surface is rendered free from bacteria by the same procedure.

We, therefore, take the liberty of recommending the avoidance of severe crushing to accomplish a successful result. Furthermore, it does not seem wise, in an effort to accomplish an aseptic procedure, to sacrifice a technique by exposing it to failure of hemostasis. All our observations account for this reasoning.

SUMMARY

Lately the surgeons are divided in their choice of one of the two techniques for gastrectomy: the Billroth I or II methods.

The best practice is to accept both procedures leaving the choice as a tactical decision.

By careful observation of the patient or during the course of the operation we can decide which technique may best be applied.

A gastroduodenostomy is often indicated and we have methodized its steps following the principles of the aseptic technique used in gastrojejunostomy.

The use of the Von Petz instrument, hemostasis with the electric cautery, and some special clamps constitute the basic points of the oblique and aseptic anastomosis.

The success of gastroduodenostomy has been, in our hands, equal to that of gastrojejunostomy, technically speaking.

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PRIMARY NONSPECIFIC ULCERS OF THE SMALL INTESTINE*

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THE jejunum and ileum are sometimes the seat of local nonspecific inflammatory ulcers which apparently have no relation to any other disease process. Whether or not these ulcers are manifestations of a single pathologic condition, they are readily distinguishable from other ulcerative lesions of the small intestine because they are usually small and solitary and are not accompanied by pathologic changes in the remainder of the intestine. For want of a better term they have been called "primary" or "simple" ulcers. Primary ulcers produce few symptoms until they bleed, obstruct the lumen of the intestine, or perforate into the general peritoneal cavity. Usually they are recognized only at laparotomy or at necropsy performed after one of these complications has taken place.

Primary ulcers of the small intestine were first described by Matthew Baillie in 1805. Cruveilhier,[1] when writing about gastric ulcers in 1830, remarked that similar "simple" ulcers had been found in the jejunum and ileum. The first review of the subject was made by Combes,[2] who collected reports of thirty-six cases of primary ulcer of the small and large intestine in 1897. Since that time numerous reports have appeared, among which are the reviews by Morrin[3] and Ebeling.[4]

This paper presents a review of the reports of forty-five recently occurring cases of primary jejunal and ileal ulcers. It includes a review of all the reports of cases available in the literature since the appearance of Morrin's article in 1931, a series of thirty-one cases (Table I), and, in addition, a review of the records of all proved cases which have been encountered at the Mayo Clinic, a series of fourteen cases (Cases 1 to 14). Nine of the fourteen cases included in the latter series were reported by Brown and Pemberton[5] in 1936.

PATHOLOGY

A practical definition, in pathologic terms, of primary ulcer has been given by Grasmann.[6] He stated that a simple (or primary) ulcer is a defect of the substance of the mucosa and often of the deeper layers of the small intestine. It is usually localized or single, with well-defined boundaries and with little or no surrounding inflammatory reaction. The etiology of the ulcer, whether acute or chronic, is unknown and its pathogenesis is uncertain.

The ulcer has a fairly characteristic appearance pathologically. Typically the primary ulcer is rounded, with a smooth base, a so-called punched-out

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TABLE I. SUMMARY OF CASE REPORTS PUBLISHED SINCE MORRIN'S REVIEW IN 1931; THIRTY-ONE CASES (EXCLUSIVE OF MAYO CLINIC CASES)

AUTHOR	DATE	AGE (YR.) AND SEX	ULCER		COMPLICATION	SYMPTOMS AND THEIR DURATION	LABORATORY FINDINGS	POSSIBLE PREDISPOSING FACTORS	TREATMENT	RESULT
			LOCATION	NUMBER						
Gutierrez*	†	30, M	Jejunum	1	Perforation	Sudden onset; severe abdominal pain			Laparotomy, perforation not found	Died after 4 days
Bigger[19]	1926†	20, M	Ileum, 3 in. (7.6 cm.) proximal to valve, antimesenteric	1	Perforation	1 wk.: fatigability; sudden perforation	Wassermann negative		Excision of ulcer	Recovery
Bigger	1926†	43, M	Ileum, junction of upper and middle thirds	1	Perforation	Sudden onset; severe abdominal pain and vomiting			Excision of ulcer, enterostomy	Died after 4 days
Heinatz[20]	1928†	24, M	Jejunum, 60 cm. distal to flexure	1	Perforation	1 yr.: sour eructations and periumbilical pain 3 to 4 hr. after meals, relief by fasting; sudden perforation		Healed duodenal ulcer and active gastric ulcer	Operation	Died after 11 days
Zeno, Santorelli, and Ercole*	1931	21, F	Jejunum, 80 cm. distal to flexure, antimesenteric	1	Perforation	Abdominal pain and diarrhea; sudden perforation			Resection and end-to-side anastomosis	Died after 4 days
Lind[7]	1931	52, M	Ileum, 18 in. (45 cm.) and 28 in. (71 cm.) proximal to valve, free border	2	Perforation	8 mo.: distress after eating; 4 mo.: diarrhea; sudden perforation			Laparotomy, perforation not found	Died after 8 hr.
Babington[8]	1932	50, M	Ileum	Several	Obstruction	15 yr.: bouts of severe abdominal pain and vomiting	Wassermann negative	Giardia lamblia in intestine	Resection and lateral anastomosis	Recovery
Moiroud[21]	1932	35, M	Ileum, 60 cm. proximal to valve, free border	1	Perforation	Sudden severe abdominal pain while lifting	Typhoid agglutination negative	Effort	Closure of perforation	Died
Ebeling[14]	1933	36, M	Jejunum, 6 in. (15 cm.) distal to flexure, annular	1	Hemorrhage, obstruction	6 yr.: indigestion; 4 yr. before admission: episode of melena; 8 wk.: abdominal pain and vomiting	Wassermann negative; obstruction to barium meal		Resection and lateral anastomosis	Recovery
Zaffagnini*	1933	23, M	Jejunum, free border	1	Perforation	Those of sudden perforation			Operation	Died after few hours
				1	Perforation	Sudden severe perium-		Vascular?	Laparot-	Died after

Zaffagnini*	1933	23, M	Jejunum, free border	1	Perforation	Those of sudden perforation			Operation	Died after few hours
Adamek[22]	1934	61, F	Jejunum, distal portion	1	Perforation	Sudden severe periumbilical pain		Vascular?	Laparotomy, perforation not found	Died after 4 days
Desyacques and Doucet-Bon*	1935	45, M	Jejunum, 10 cm. distal to flexure, antimesenteric	1	Perforation, hemorrhage	Several mo.: dyspepsia; hematemesis; sudden perforation			Operation	Died after few hours
Smith[18]	1935	30, M	Jejunum, 2 feet (61 cm.) distal to flexure, antimesenteric	1	Perforation, hemorrhage	2 yr.: distress after eating; sudden perforation	Wassermann negative; occult blood test positive	Buerger's disease	Closure of perforation	Recovery
Levalle and Lubetkin[23]	1936	21, F	Ileum	1	Perforation	4 wk.: periumbilical pain in attacks; vomiting; sudden perforation			Closure of perforation	Recovery
Coletti*	1936	56, M	Jejunum, free border	1	Perforation, hemorrhage	2 yr.: vague generalized abdominal pain preceding defecation; 2 mo. before admission: severe rectal hemorrhage; sudden perforation			Excision of ulcer	Recovery
Davidovitch*	1937	23, M	Jejunum	1	Perforation	Severe abdominal pain for 3 days			Closure of perforation	Recovery
Lastra[24]	1937	47, M	Jejunum, 4 cm. distal to flexure	1	Localized perforation onto transverse colon causing adhesions, obstruction, hemorrhage	4 mo.: periumbilical pain; intermittent intestinal obstruction	Wassermann negative; occult blood test positive		Resection and end-to-end anastomosis	Recovery
Baravalle[25]	1937	37, M	Ileum, proximal portion, free border	1	Perforation	4 or 5 yr.: periodic abdominal pain 4 hr. after eating, relieved by food; sudden perforation			Closure of perforation	Recovery
Masciottra[26]	1938	56, F	Jejuno-ileal region, close to mesentery	1	Perforation	2 days: 2 episodes of severe abdominal pain, the second accompanied by symptoms of perforation		Infection of upper part of respiratory tract	Resection and end-to-end anastomosis	Recovery
Pierangeli[27]	1938	57, M	Ileum, 10 cm. proximal to valve, free border	1	Perforation	Those of sudden perforation	Wassermann negative		Closure of perforation	Recovery

*The case reports of Gutierrez, Zeno and co-workers, Zaffagnini, Desyacques and Doucet-Bon, Coletti, and Davidovitch were abstracted and quoted by Mangione.

†Reported previous to 1931 but not included in Morrin's review.

TABLE I.—(CONT'D)

AUTHOR	DATE	AGE (YR.) AND SEX	ULCER		COMPLICATION	SYMPTOMS AND THEIR DURATION	LABORATORY FINDINGS	POSSIBLE PREDISPOSING FACTORS	TREATMENT	RESULT
			LOCATION	NUMBER						
Mangione[28]	1938	46, F	Jejunum, close to flexure	1	Perforation, fistula	1 mo.: episodes of severe abdominal pain, diarrhea; mass in abdomen	Wassermann negative; occult blood test positive		Lateral anastomosis proximal to fistula	Died after 3 wk.
Tosonotti[29]	1940	15, M	Ileum, 20 cm. proximal to valve, free border	1	Perforation	Those of sudden perforation	Wassermann negative	Pleurisy with effusion	Closure of perforation	Recovery
Segelman[30]	1940	40, M	Ileum	1	Perforation	10 days; abdominal pain and fever; diagnosis: peritonitis			No operation	Died
Segelman	1940	31, M	Ileum, 25 cm. proximal to valve	1	Perforation	Those of sudden perforation	Wassermann negative; typhoid agglutination negative		Closure of perforation	Recovery
Segelman	1940	32, M	Ileum, 50 cm. proximal to valve	1	Perforation	10 days: malaise; sudden perforation			Resection and end-to-end anastomosis	Recovery
Robinson and Wise[15]	1940	52, F	Ileum, 15 in. (38 cm.) proximal to valve, antimesenteric	1	Perforation	1 mo. before admission: two episodes of periumbilical pain; sudden perforation		Heterotopic mucosa	Resection and lateral anastomosis	Died after 14 days
Robinson and Wise	1940	20, M	Ileum, middle third	1	Perforation	Those of sudden perforation			No operation	Died
Piulachs[9]	1941	46, M	Ileum, 20 and 50 cm. proximal to valve, free border	2	Perforation	Those of sudden perforation	Typhoid culture and agglutination negative		Resection and lateral anastomosis	Died after 1 day
Dowdle[10]	1942	59, M	Jejunum, 1 in. (2.5 cm.) distal to flexure, annular	2	Obstruction, hemorrhage	10 yr.: black stools; progressively severe vomiting after meals for 6 mo.	Obstruction to barium meal		Resection and lateral anastomosis	Died after 8 days
Chambers[11]	1943	34, F	Jejunum, 3 in. (7.6 cm.) distal to flexure	4	Hemorrhage	6 mo.: colicky abdominal pain; malnutrition; massive fatal intestinal hemorrhage		Tuberculosis?	No operation	Died
Marqués and Quintana[31]	1944	46, M	Jejuno-ileal region, free border	1	Perforation	Sudden perforation; lifting at time of onset of symptoms		Effort	Closure of perforation	Recovery

margin (Fig. 1), and little induration of the surrounding tissues. The serosa beneath the ulcer is whitened or reddened. The intestine is frequently narrowed by the fibrous scarring of the ulcer. When examined microscopically, the ulcer is found to have a base which does not undermine the near-by epithelium and which is covered by a thin layer of fibrin and leucocytes. The underlying inflammatory reaction involves all the layers of the intestine but does not extend far beyond the margin of the ulcer laterally. The ulcer base consists of a layer of granulation tissue which replaces the muscularis mucosae, is infiltrated by lymphocytes and plasma cells near the surface, and is underlaid by a varying amount of mature collagenous connective tissue (Figs. 2 and 3). Rarely extensive accumulations of lymphoid tissue are found in the wall of the bowel. The epithelium at the margin of the ulcer shows the changes characteristic of active proliferation, namely, small darkly stained cells with irregularly arranged nuclei and many mitotic figures. The remainder of the tissues either are normal or show minor degrees of inflammation. Enlargement of regional lymph nodes is unusual. Acute ulcers, in which little or no fibrosis has occurred, have been described but chronic ulcers are more common, even in cases in which the clinical illness has been of short duration.

Fig. 1 (Case 13).—Portion of a primary jejunal ulcer; the punched-out margin of the ulcer is demonstrated.

Primary ulcers have been found throughout the small intestine. Of all the known cases (including those collected by Morrin), in 38 per cent the ulcer was in the jejunum and in 62 per cent in the ileum. Of the forty-five recently occurring cases, the ulcer in eleven was less than 80 cm. from the duodeno-jejunal flexure and in nineteen it was less than 60 cm. from the ileocecal valve. Brown and Pemberton commented on the frequency with which the lesion was found close to the ileocecal valve. It would also appear that the lesion is frequently found a short distance below the duodenojejunal flexure (Table II). A primary ulcer does not have any constant relation to the mesentery.

Although the primary ulcer is typically solitary as contrasted to other types of ulcers, nonspecific ulcers with a characteristic pathologic appearance have been found in localized groups. Grasmann collected reports of four cases in which multiple ulcers occurred simultaneously; Lind,[7] Babington,[8] Piulachs,[9] Dowdle,[10] and Chambers[11] have each reported cases since then, and in the Mayo Clinic series there was one such case (Case 10). When the lesions are multiple, a group of two or three is the rule.

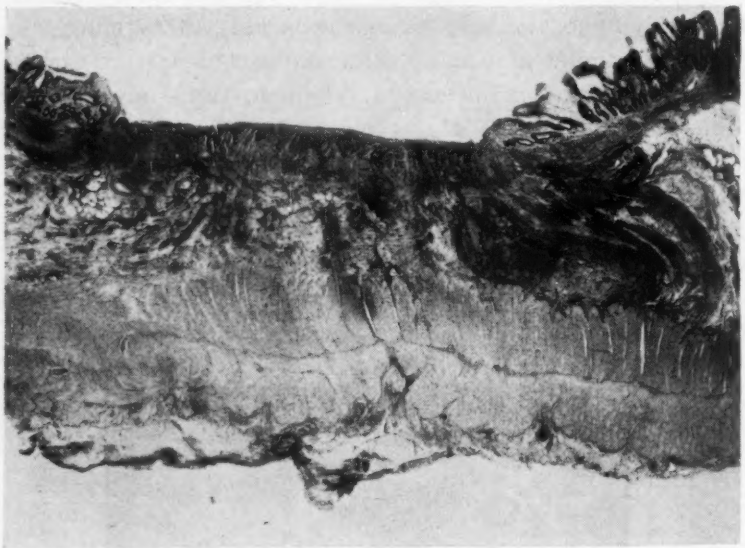

Fig. 2 (Case 5).—Primary ileal ulcer. Note well-defined margins, narrow superficial zone of necrosis, underlying zone of granulation tissue infiltrated by inflammatory cells, and deeper zone of fibrosis (hematoxylin and eosin, ×7½).

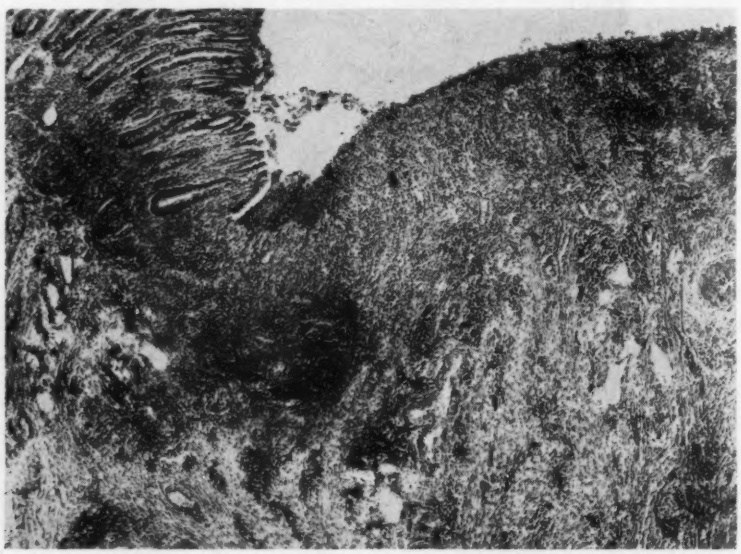

Fig. 3 (Case 13).—Primary jejunal ulcer. The granulation tissue is densely infiltrated with inflammatory cells near the surface but is blended with young connective tissue peripherally. The inflammatory cells are mostly plasma cells and lymphocytes, with a few polymorphonuclear leucocytes (hematoxylin and eosin, ×25).

TABLE II. LOCATION OF PRIMARY ULCERS IN THE JEJUNUM AND ILEUM (FORTY-FIVE CASES)

LOCATION	CASES
Jejunum	
Proximal 80 cm.	11
Unspecified	4
Middle and distal portions	2
Jejuno-ileal region	3
Ileum	
Upper and mid-portions	3
Unspecified	3
Terminal 60 cm.	19
Total	45

Primary ulcers are potentially dangerous because of the complications which may develop. In every reported case of ulcer which has caused clinical symptoms or which has been recognized at laparotomy, perforation, bleeding, or obstruction has occurred. Perforation occurred in 81 per cent of all recorded cases, bleeding in 15 per cent, and clinical obstruction in 9 per cent; from these figures it is apparent that a few patients suffered from multiple complications (Table III).

TABLE III. COMPLICATIONS IN 130 CASES

COMPLICATION	85 CASES BEFORE 1931		31 CASES 1931 TO 1944		14 CASES FROM MAYO CLINIC		TOTAL, 130 CASES	
	NUMBER	PER CENT	NUMBER	PER CENT	NUMBER	PER CENT	NUMBER	PER CENT
Perforation	75	88	27 (25 free, 2 localized)	87	3 (1 free, 2 localized)	21	105	81
Bleeding	2	2	8 (5 gross, 3 occult)	26	9 (7 gross, 2 occult)	64	19	15
Obstruction (clinical)	5	6	3	10	4	28	12	9

Perforation, which resulted most often in generalized peritonitis, was found in thirty of the forty-five recently occurring cases. In four cases, however, the perforation had been localized by inflammatory adhesions; two of these localized perforations were associated with inflammatory abdominal masses, one was associated with a jejunocolic fistula, and one was surrounded by extensive adhesions to the mesentery.

Recurrent, prolonged, and occasionally massive bleeding has been associated with primary ulcer. In seventeen of the forty-five cases just referred to, unmistakable clinical signs of loss of blood were present; in twelve of these seventeen, grossly visible blood had passed from the gastrointestinal tract. Five of the ulcers which bled were annular, two were located near the mesentery, and five were at a distance from the mesentery. In every case in which an annular ulcer was reported, there was a history of recognizable gastrointestinal bleeding.

Symptoms of intestinal obstruction were recorded in seven of the forty-five cases referred to previously. Clinical evidence of obstruction was considerably less frequent than was the pathologic finding of dilatation of the intestine proximal to a stricture at the site of the ulcer. This is well demonstrated in the series of fourteen cases studied. In nine of these cases there was pathologic

evidence of dilatation of the bowel proximal to a stenotic ulcer, but in only four of the nine was there sufficient stenosis to cause clinical symptoms of obstruction.

Recurrence of primary ulcer has been reported. The patient of Gale[12] had three different episodes of perforation, each of which followed an attack of furunculosis. Fischer[13] and also Brown and Pemberton[5] (Case 5, reported herein) have reported cases in which clinical symptoms recurred after resection for ulcer.

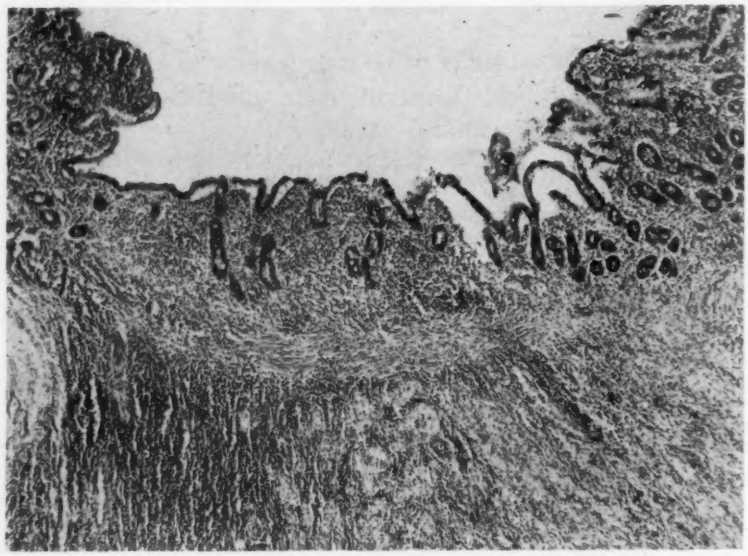

Fig. 4, (Case 11).—Primary jejunal ulcer. The surface is covered by a thin layer of mucous membrane which is not thrown into villi. The presence of well-organized granulation tissue and fibrosis beneath the ulcer indicates a process of healing (hematoxylin and eosin ×40).

From available facts, it cannot be said whether a primary ulcer of the type herein described ever occurs without producing clinical manifestations or whether an ulcer ever heals spontaneously. It would seem possible that an ulcer might occur without bringing about symptoms necessitating laparotomy and, therefore, without ever being detected. An ulcer of this description has not been reported as an incidental finding at necropsy. There is good presumptive evidence that an ulcer may heal spontaneously. For example, Oudard and Jean[14] reported the case of a patient who had a group of ulcers, one of which seemed to be in the healing stage. The appearance of the ulcer microscopically in Case 11 reported herein, in which the base of the ulcer consisted of an abnormally thin layer of mucous membrane covered by epithelium and beneath which were granulation tissue and fibrosis, suggests healing (Fig. 4). Also, it is known that many patients whose perforated ulcer is treated only by simple closure have no more symptoms.

ETIOLOGY

The cause of primary ulcer is not known. The occurrence of this type of ulcer has been attributed to infection, peptic digestion, trauma, or vascular ab-

normalities. No infectious agent has been found in constant association with the lesion. Syphilis and tuberculosis have been excluded as possible etiologic agents in most cases. A certain number of high jejunal ulcers have been found in association with peptic ulcers, but many more have not. The widely quoted theory that heterotopic gastric mucosa in the intestine might contribute to the formation of these ulcers, just as such tissue contributes to the formation of ulcers about Meckel's diverticulum, cannot be substantiated because microscopic amounts of gastric tissue have almost never been found[15] in association with these ulcers. At times perforation of chronic ulcer has been associated with external trauma.[16] Only rarely has an intraintestinal foreign body been found near an ulcer. Cornioley's[17] suggestion that all the ulcers are secondary to ischemia produced by localized arteriolosclerosis or embolism has not been generally accepted. In the cases studied there was no evidence of heterotopic gastric mucosa or of any vascular abnormality. Thorough discussions of etiologic theories may be found in the papers of Ebeling and of Oudard and Jean.

Oudard and Jean have concluded that these ulcers might well result from a variety of causes, for they have been associated with distinctly different diseases.

Although some of the so-called primary ulcers have probably in actual fact been caused by infections, trauma, or peptic digestion, the pathogenesis of most primary ulcers must remain obscure.

OCCURRENCE

Primary ulcer of the jejunum and ileum is rare. Morrin collected reports of eighty-five cases in the literature up to 1931, and to this number forty-five cases have since been added, making a total of 130 cases. The number of cases studied at the clinic is equivalent roughly to one per 100,000 patients registered. In ninety-seven of the known cases the patients were men, and in thirty-three they were women, a ratio of about 3:1. The youngest patient who was reported to have such a lesion was 1 year old, and the oldest, 77; the majority were adult persons. The average age of all patients was 43 years.

SYMPTOMS AND LABORATORY FINDINGS

The symptoms of primary ulcer depend chiefly on complications. For convenience of discussion, known cases may be divided into two groups according to whether free perforation did or did not occur. When free perforation occurred, the symptoms were those characteristic of any sudden perforation of a viscus into the peritoneal cavity. In contrast to gastric or duodenal perforation, jejunoileal perforation was often associated with a point of maximal tenderness to the left of the umbilicus in the reports of cases studied.[18] The perforations of primary ulcer were preceded by some sort of chronic abdominal pain in about one-half of the reported cases.

The exact diagnosis of chronic nonperforated primary ulcer has seldom been made clinically. Under certain circumstances, however, the symptoms may suggest a lesion of the small intestine. Primary ulcer has been found at laparotomy done for (1) episodes of crampy abdominal pain and vomiting sug-

gestive of obstruction of the small intestine, (2) anemia and unexplained gastrointestinal bleeding, and (3) postprandial epigastric or periumbilical pain. In certain cases the abdominal symptoms have been so vague or so mild as to defy diagnostic classification.

In addition to clinical findings, those laboratory tests which are of specific aid in establishing a diagnosis of chronic jejunoileal ulcer depend on the fact that the ulcer may bleed or cause obstruction. Benzidine and guaiac tests for occult blood in the stool have proved to be of considerable value. In several cases a history of melena was confirmed by the results of a test for occult blood; in five of the seventeen cases, referred to previously, in which bleeding occurred, the result of the test for occult blood was the only positive evidence of bleeding. No case of primary ulcer has yet been reported in which such a test, when done, gave negative results. Brown and Pemberton, Smith and Ebeling have called attention to the value of this test as a diagnostic measure. Roentgenologic demonstration of a small intestinal ulcer is rarely possible. However, on two occasions constricting ulcers have been demonstrated by means of a barium meal.[4, 10] In the series studied herein, a constricting ulcer could not be demonstrated in any of the four patients to whom a barium meal was administered.

DIAGNOSIS

Because of the rarity of primary ulcer and the variety of clinical symptoms which it may produce, the clinical diagnosis was so obscure that it was established in only one reported case.[4] However, it is useful to think of the possibility of occurrence of these lesions in the following groups of patients: (1) patients who present a problem of gastrointestinal hemorrhage, acute or chronic, in whom there is no roentgenologic evidence of abnormality of the esophagus, stomach, duodenum, or colon; (2) patients who have symptoms and signs of peritonitis suggestive of a ruptured peptic ulcer, in whom no gastric or duodenal perforation is discovered at laparotomy, and (3) patients who present a problem of recurrent postprandial pain suggestive of peptic ulcer, in whom there is roentgenologically no evidence of gastric or duodenal ulceration.

MORTALITY RATE

The mortality rate in cases of primary ulcer is high. Morrin found reports of fifty-six fatalities among the records of eighty-five cases (66 per cent). There were twenty-one deaths among the forty-five recently occurring cases, a mortality rate of 47 per cent. In the latter series of cases, ulcers, associated with free perforation and generalized peritonitis accounted for fourteen deaths in twenty-six cases (54 per cent), and ulcers associated with local perforation or not associated with perforation accounted for seven deaths in nineteen cases (37 per cent). Among twelve patients of the recently reported series who had grossly recognizable intestinal bleeding, one had a fatal hemorrhage.

TREATMENT

The treatment in all cases in which the lesion was recognized during life was surgical (Table IV). In most cases the gravity of the complications was

such that laparotomy had been done without specific preoperative diagnosis. Robinson and Wise have concluded that "the simplest procedure compatible with the condition encountered . . . should be done." In the presence of free perforation, transverse closure of the perforation would seem to be the best procedure, according to the results obtained in the twenty-six cases of the recently occurring series in which this complication was present. Nine of those twenty-six patients were treated by simple closure of the perforation, with one fatality; three were treated by excision of the ulcer and transverse closure of the bowel, with one fatality, and five were treated by resection and anastomosis, with three fatalities. The three patients whose perforated ulcers were not discovered at the time of laparotomy and the three patients who did not undergo operation all died. Three other patients died after unspecified surgical procedures.

TABLE IV. MORTALITY IN RELATION TO SURGICAL OR NONSURGICAL TREATMENT (FORTY-FIVE CASES)

COMPLICATION	TOTAL CASES	TREATED SURGICALLY		NOT TREATED SURGICALLY	
		CASES	DEATHS	CASES	DEATHS
Free perforation	26	23	11	3	3
No perforation or localized perforation	19	18	6	1	1 (died of hemorrhage)
Total	45	41	17	4	4

More extensive procedures have been used in the treatment of ulcer which has not perforated. Of eighteen such patients who underwent surgical treatment, one was treated satisfactorily by simple closure of a local perforation, three were treated, with one fatality, by lateral anastomosis without resection, and fourteen were treated by resection and anastomosis, with five fatalities. Removal of the ulcer would not appear to be strictly necessary, since those patients in whom the ulcer was not actually removed and those in whom a simple closure was done for perforation all had little further trouble. The occasional tendency to recur and the difficulty of excluding from consideration the possibility of malignancy on the basis of gross appearance, however, would suggest that resection of the diseased tissue would be the procedure of choice whenever such procedure would not materially increase the risk of operation.

REPORT OF CASES

In view of the rarity of primary ulcer the records of all the cases of primary ulcers encountered at the Mayo Clinic have been reviewed. The first nine cases in this series, which were reported in abstract by Brown and Pemberton in 1936, have been included in more detail. Five new reports of cases complete the series. These cases have been selected in accordance with the pathologic criteria stated by Grasmann. Those ulcers which seemed to be acute and which were found incidentally in cases in which death had resulted from other diseases were not included in this series. In the majority of cases reported in the literature, the complication of acute perforation was present, whereas in virtually all the cases studied at this institution, chronic complications led to

the diagnosis, possibly because comparatively few emergent abdominal conditions of any type were seen here. The deaths which followed surgical treatment in this series of cases all occurred before the year 1933.

CASE 1 (Brown and Pemberton, Case 1).—An architect, 58 years old, had had recurrent attacks of severe colicky periumbilical pain for three years. The pain lasted hours at a time and recurred every few months. During the ten months before admission to the clinic, the pain had become more frequent and had been brought on by eating. The patient had lost forty-five pounds (20.4 kilograms).

The abdomen was distended and on its surface peristaltic waves could be seen. A mass was palpable in the left lower quadrant. The other findings were not significant.

At laparotomy an inflammatory ulcer was found in the distal part of the ileum. At the site of the ulcer there was an obstructing stricture and proximally there was distention of the bowel. A lateral anastomosis was performed and the site of the ulcer was not disturbed. Convalescence was uneventful. The patient was reported to be in good health six years later.

CASE 2 (Brown and Pemberton, Case 2).—A housewife, 58 years old, had had vague abdominal pains as well as numerous other complaints for many years. Three weeks before admission she began to have griping periumbilical pain accompanied by bloating of the abdomen and borborygmi.

The abdomen was somewhat tender and peristalsis could be seen on its surface. The other findings were not significant.

At laparotomy an obstructing ulcer was found 100 cm. proximal to the ileocecal valve. The mesentery beneath the ulcer was involved in an inflammatory reaction and free fluid was present in the peritoneal cavity. A lateral anastomosis was established and the site of the ulcer was not disturbed. Convalescence was uneventful but during the remaining eleven years of her life the patient continued to complain of vague abdominal pain.

CASE 3 (Brown and Pemberton, Case 3).—A janitor, 47 years old, had had dull, burning epigastric pain for three years. The pain began about one hour after eating, was relieved by so-called dyspepsia pills, and was occasionally accompanied by a sensation of bloating. During the three weeks before admission the pain had become more severe and vomiting had occurred frequently.

The patient appeared ill and was evidently in pain. Tenderness and rigidity were present in the midepigastric region.

At laparotomy two or three loops of bowel were found to be adherent about an abscess which communicated with a perforating ulcer of the ileum. The ulcer was 5 cm. proximal to the ileocecal valve. A few small edematous ecchymotic spots were seen in the near-by ileum. The perforation in the ileum was closed. Postoperatively an enterocutaneous fistula developed but the abdominal symptoms had been relieved for at least a year when the patient was last seen.

CASE 4 (Brown and Pemberton, Case 5).—A carpenter, 47 years old, had passed massive tarry stools many times during the eight years before admission. He had no gastrointestinal symptoms except occasional sensations of bloating. During the last four months before admission, he had become extremely weak.

The spleen was palpable. The hemoglobin measured 4.4 Gm. per 100 c.c. of blood. The results of guaiac tests were repeatedly positive for occult blood in the stool.

At laparotomy an ulcer which had produced a partially obstructing stricture was found halfway between the duodenum and cecum. The lesion was removed and an end-to-end anastomosis and a proximal enteric stoma were established. The ulcer was annular, measured 6 mm. in cross section, and was atypical in that the adjacent submucosa was densely infiltrated by lymphocytes. Five years afterward the patient was reported to be in good health.

CASE 5 (Brown and Pemberton, Case 6).—A grocer, 35 years old, complained of intermittent abdominal pain which had been present in increasing severity for six years. The pain

was located in the upper part of the abdomen and was sometimes brought on by eating. There were sensations of abdominal bloating and anorexia. He had lost twenty-five pounds (11.3 kilograms).

The patient looked ill but no physical abnormalities were found. The value for the hemoglobin was 7.3 Gm. per 100 c.c. of blood, and the results of guaiac tests were repeatedly positive for occult blood in the stool.

At laparotomy an obstructing ileal ulcer with a punched-out appearance was found 90 cm. proximal to the ileocecal valve. The involved part of the ileum was resected, an end-to-end anastomosis was established, and a proximal enteric stoma was made. The ulcer measured 1.5 cm. in diameter and had a typical appearance pathologically as shown in Fig. 2.

One and one-half years later the patient again noticed sharp pain to the left of the umbilicus, which appeared about one-half hour after meals and was accompanied by belching. The patient was last seen two years after operation, at which time he had the same symptoms and was still anemic. Because of the similarity of the symptoms to those which occurred before operation, the patient was considered to have a recurrence of the ulcer.

CASE 6 (Brown and Pemberton, Case 8).—A laborer, 26 years old, had had increasingly severe abdominal pain for one and one-half years. The pain was located across the middle part of the abdomen, it sometimes extended to the back, and it began about one-half hour after eating. Vomiting occasionally relieved the pain. The patient had lost ten pounds (4.5 kilograms).

Physical findings were not significant. The hemoglobin was 30 per cent of normal and the results of guaiac tests were positive for occult blood in the stool.

At laparotomy a solitary ulcer was found near the mesentery of the ileum about 2 feet (61 cm.) proximal to the ileocecal valve. The mesentery beneath the ulcer was considerably thickened. The lesion was removed and a lateral anastomosis was established. The patient died four days later because of general peritonitis. The ulcer was rounded, measured 2.5 cm. in diameter, and had a typical appearance pathologically.

CASE 7 (Brown and Pemberton, Case 10).—A housewife, 24 years old, had been severely anemic most of the time for ten years. At intervals during this period she had been troubled by constipation and by severe cramps across the lower part of the abdomen. During the last three months before admission, she had passed tarry or bloody stools on several occasions and had had severe abdominal cramps accompanied by vomiting. She had lost twenty-three pounds (10.4 kilograms).

The results of physical examination were not remarkable. The content of hemoglobin was 7.4 Gm. per 100 c.c. of blood, and the results of guaiac tests were positive for occult blood in the stools.

At laparotomy a constricting ulcer of the ileum 45 cm. proximal to the ileocecal valve was found. The lesion was removed and an end-to-end anastomosis and a sidetracking lateral anastomosis were established. Three weeks later the patient died because of general peritonitis. The ulcer was annular and had a characteristic appearance microscopically.

CASE 8 (cited by Brown and Pemberton).—A farmer, 46 years old, had been pale, weak, and easily fatigued for eight months. After examination a diagnosis of pernicious anemia was established. As a phase of a method of treatment in vogue nearly thirty years ago, ileosigmoidostomy was performed. During the operation an ulcer was found in the ileum 15 cm. proximal to the ileocecal valve. The ulcer was removed. It measured 1.5 by 1 cm. and had the microscopic appearance of a simple ulcer. The patient died six days afterward because of general peritonitis.

CASE 9 (cited by Brown and Pemberton).—An insane patient, 47 years old, who was suffering from general paresis, complained of abdominal pain and became ill. Within a few hours he had a temperature of 104° F. The following day the patient died. At necropsy general peritonitis and a clean-cut perforation secondary to an acute nonspecific inflammatory ulcer of the ileum, 80 cm. proximal to the ileocecal valve, were observed.

CASE 10.—A housewife, 45 years old, had been troubled by constipation for four years and had lost twenty pounds (9.1 kilograms) because of a self-imposed diet. For eight

months she had noticed increasingly severe, diffuse abdominal pain which was relieved by defecation and which sometimes awakened her at night. She had had some nausea and a sensation of abdominal fullness after eating.

The abdomen was tympanitic and tender about the umbilicus. The value for hemoglobin was 68 per cent. Other studies, including urinalysis, a flocculation test for syphilis, and roentgenography of the stomach and colon, did not give significant results.

During a period of observation the patient continued to have abdominal pain and the value for hemoglobin fell to 58 per cent.

At laparotomy two constrictions secondary to punched-out nonspecific ulcers were found 8 cm. apart about 30 cm. proximal to the ileocecal valve. One ulcer was annular and the other was oval; the diameter of each varied from 1 to 2.5 cm. The appearance microscopically was similar to that of other primary ulcers previously described. The mesenteric lymph nodes were enlarged but the remainder of the intestine appeared normal. The involved segment of bowel was resected and an end-to-end anastomosis was established. Five days later the patient died of general peritonitis.

CASE 11.—A Negro, 65 years old, who had been troubled by constipation for many years, had noticed crampy midabdominal pain for two months. The pain was intermittent, often became worse fifteen minutes after meals, and sometimes awakened the patient at night. He lost twenty pounds (9.1 kilograms).

The results of physical examination were not remarkable. The content of hemoglobin was 7.0 Gm. per 100 c.c. of blood and the result of a benzidine test was positive for small amounts of occult blood in the stool. Other studies, including urinalysis, a flocculation test for syphilis, and roentgenologic examination of the stomach, small intestine, and colon, gave essentially negative results.

At laparotomy an ulcer 1 cm. in diameter was found in the midjejunal region, away from the mesentery. The involved segment of bowel was excised and an end-to-end anastomosis was established. Convalescence was uneventful. The appearance of the ulcer microscopically suggested a process of healing, as described previously in this paper (Fig. 4).

CASE 12.—A man, 55 years old, had had four episodes of massive gastrointestinal bleeding in twelve years. On one occasion the bleeding had been manifested as hematemesis, on other occasions, as melena. He had had no abdominal symptoms save an occasional so-called gassy sensation after eating.

The findings on physical examination were not significant. The value for hemoglobin was 11.7 Gm. per 100 c.c. of blood. On roentgenoscopic examination changes suggestive of a duodenal ulcer were seen. The results of urinalysis and a flocculation test for syphilis were essentially negative.

At laparotomy no external evidence of duodenal abnormality was encountered but a chronically obstructing lesion of the jejunum about 20 cm. distal to the duodenojejunal flexure was found. The lesion was an ulcer 2 by 2.5 cm. in diameter, located adjacent to the mesentery and associated with three inflammatory lymph nodes. On microscopic examination a dense collection of lymphocytes and plasma cells beneath the ulcer, with edema of the adjacent submucosa, was seen. The involved segment of bowel was resected and an end-to-end anastomosis was established. Convalescence was uneventful.

CASE 13.—For six years a housewife, 31 years old had had epigastric pain which often began two hours after eating and which was relieved by drinking milk. The pain was present only at irregular intervals. The patient had passed bulky tarry stools and had become weak on ten different occasions. The patient's father was said to have suffered from a stomach or peptic ulcer.

Save for slight epigastric tenderness there were no significant findings on physical examination The content of hemoglobin was 9.7 Gm. per 100 c.c. of blood. Other tests, including urinalysis, a flocculation test for syphilis, tests for coagulation of blood and bleeding time, and roentgenoscopy of the stomach, small intestine, and duodenum, gave negative results.

At laparotomy an indurated constricting lesion was found in the jejunum 45 cm. distal to the duodenojejunal flexure. This lesion was removed and was found to contain an ulcer 1.5 by 2 cm. in diameter, located 1 cm. from the mesentery. Microscopically the ulcer was similar to those described previously (Fig. 3). There was no lymphadenopathy. An end-to-end anastomosis was established and the patient had an uneventful convalescence.

CASE 14.—A housewife, 60 years old, had had abdominal cramps which lasted one or two days at a time, for two years. One week before admission she became unable to move the bowels and began to have crampy abdominal pain and distention. The latter symptoms progressed and she vomited foul brown material.

The patient was a thin sick-looking woman whose abdomen was moderately distended and tympanitic. On roentgenographic examination multiple distended loops of small intestine were seen.

After nasogastric intubation the patient improved temporarily. On the sixth day in the hospital laparotomy was performed. The intestine was obstructed by adhesions about a perforating ulcer located in the terminal part of the ileum. There was no lymphadenopathy. The lesion was removed and an end-to-end anastomosis was made. The ulcer was similar to those seen in the other cases save for a more acute inflammatory process. The patient's convalescence was satisfactory.

SUMMARY

Nonspecific localized ulcerations of the jejunum and ileum are so similar pathologically as to justify their classification as a group under the name "primary" or "simple" ulcers. Although the lesions are characteristically solitary, small groups of ulcers are sometimes found. The etiology of primary ulcers is unknown. There is little direct evidence to support the theories that they are caused by infection, irritation from gastric secretions, trauma, or vascular abnormalities.

The symptoms of primary ulcer are for the most part secondary to the complications of perforation, bleeding, or obstruction. The possibility of these lesions should be considered in the presence of unexplained intestinal bleeding or of peritonitis which suggests acute visceral perforation when such perforation cannot be found in the stomach or duodenum.

The mortality rate in patients suffering from primary ulcer is high. The lesion has been recognized during life only after some complication has led to surgical intervention.

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CARCINOMA OF THE GALL BLADDER

Report of Seventy-five Cases

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ALTHOUGH numerous reports concerning the subject of carcinoma of the gall bladder have emphasized the gloomy outlook in this disease, it seems justifiable to continue the analysis of large series of cases in order to arrive at some more promising active or prophylactic therapy. Reports of cure of carcinoma of the gall bladder are indeed rare. Mohardt,[1] in a comprehensive analysis of the literature, found a paucity of five-year cures, and stated that the survival rate, including all cases reported, varied from 0 to 6 per cent.

The contention by many physicians and surgeons alike that this disease is too uncommon to cause grave concern, is not borne out by the surveys made in recent years. In large series of consecutive autopsies, the percentage of occurrence of carcinoma of the gall bladder is shown to be .41,[2] .27,[3] and .33.[4] These autopsies, however, are unselected and include all age groups as well as patients who may have been operated upon previously. Thus, Graham[5] stated that carcinoma of the gall bladder constitutes 8 to 10 per cent of all carcinomas and Kirshbaum and Kozoll[2] placed the incidence at 3 per cent of carcinomas found in 13,300 autopsies.

Even more important is the frequent occurrence of carcinoma of the gall bladder in association with cholelithiasis. Graham found that in 8½ per cent of all cases of cholelithiasis, discovered at autopsy, carcinoma of the gall bladder was also present, and he quoted previous authors as finding an incidence of 5 to 14 per cent. In a more recent report,[6] figures of 4, 5, and 6 per cent were given. Warren and Balch[7] gave the incidence of carcinoma found at operation on such diseased gall bladders as 1 to 2½ per cent. As Mohardt indicated, however, the low incidence found in many recent reports on operative findings may in a large part be due to the fact that many more cholecystectomies are now performed on younger patients. It is evident, therefore, that cancer of the gall bladder is not as rare as some reports would have one believe.

REPORT OF CASES

In the fourteen years from 1933 to 1946, inclusive, seventy-five patients with carcinoma of the gall bladder were encountered on the ward services of the Mount Sinai Hospital. Sixty-five of these were subjected to operation, and the diagnosis was verified by microscopic examination. Of the remaining ten patients, five were moribund on admission and could not be dealt with surgically, and the other five died of unrelated disease. At necropsy, all ten were found to have carcinoma of the gall bladder. Follow-up was complete in all cases. One patient was alive thirteen and one-half years after operation; the remainder died

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within a period of thirty-five months after operation. This constitutes a five-year survival rate of 1½ per cent.

Sixty-three patients were women and twelve were men, a sex incidence of 84 and 16 per cent, respectively. This ratio of 5.2 to 1 is in accordance with that found by most observers, the average having been 4 to 1 in the collective study of Mohardt. In Table I is seen the incidence according to age. The youngest patient was 38 years old, the oldest 83 years. Over 89 per cent of the patients were between the ages of 40 and 69 years.

TABLE I. AGE INCIDENCE

AGE (YR.)	CASES	INCIDENCE (PER CENT)
30-39	1	1.3
40-49	8	10.7
50-59	29	38.7
60-69	30	40
70-79	6	8
80-89	1	1.3
Total	75	100

In an endeavor to discover some means of correlating diagnosis with the history, physical examination, and/or laboratory findings, a careful analysis of each phase of the examination was made. The past history was significant only in that thirty-two, or 42.6 per cent, had a history of one to forty years strongly suggestive of gall bladder disease, as indicated by positive cholecystograms, or because of recurrent attacks of right upper quadrant pain, fatty food intolerance, jaundice, nausea, and vomiting. The criterion of the onset of the illness in all cases, which ranged from one day to one year, was either the initial appearance of complaints or a marked transition from the longer history referable to gall bladder disease.

The predominant symptoms were as follows:

Of the total, 77.3 per cent complained of abdominal pain. This was most frequently located in the right upper quadrant, but occasionally in the epigastrium and rarely in the lower abdomen.

There was definite weight loss in 41 per cent of the patients, varying in amount from five to seventy pounds.

Jaundice was a symptom in 38.6 per cent.

Other less frequent symptoms were anorexia, nausea, vomiting, weakness, constipation, dyspepsia, diarrhea, melena, chills, and fever. Nine patients were known to have diabetes.

On physical examination the important frequent findings were a palpable mass in the right upper abdomen, 64 per cent; jaundice, 38 per cent; and a palpably enlarged liver, 28 per cent.

Laboratory diagnostic aids, such as blood counts, chemical analyses of blood and urine, and roentgen examination showed no constant aberration from the normal. The correct clinical diagnosis was made in 22.7 per cent of the patients, although its possibility was suggested in many of the others. These

clinical findings compare favorably with those found by Kirshbaum and Kozoll in a series of fifty-five cases in which the correct diagnosis was also made in 22 per cent of the patients. They related the cardinal findings to be pain, jaundice, and a palpable tumor, although these were by no means always associated. Our cardinal findings are the same, in addition to weight loss.

At operation, the findings varied from the appearance of a chronic cholecystitis to a totally inoperable neoplastic mass which in different cases involved virtually every abdominal organ. In those which were entirely inoperable, a biopsy was taken and the abdomen closed (Group I). Where it was thought that the disease was beyond surgical excision, but that some palliative procedure might prolong the life of the patient, such procedures as hepaticoduodenostomy, choledochostomy, partial cholecystectomy en bloc, excision of liver and gall bladder, or simple cholecystostomy were performed (Group II). In another group of cases it was felt by the surgeon that all visible disease could be extirpated and that the patient might possibly be cured (Group III). The remainder constitute those in which the diagnosis was made by the pathologist, and in which the surgeon at the time of operation had not suspected carcinoma (Group IV). In Table II are the average postoperative survival periods for the groups mentioned. The one living patient who is included in Group IV was operated upon thirteen and one-half years ago and is now free of any evidence of disease. This patient was 53 years old at the time of operation and she had had a three-week history indicative of acute cholecystitis. This also was the postoperative diagnosis, but the microscopic sections of the gall bladder revealed an early adenocarcinoma, as well as cholelithiasis and acute cholecystitis. If this patient is excluded, the average survival in this group becomes only eleven and one-half months, rather than thirty months.

TABLE II. SURVIVAL PERIODS

GROUP I	GROUP II	GROUP III	GROUP IV*
22 cases	27 cases	8 cases	7 cases
2.3 mo.	3.4 mo.	6.7 mo.	30.5 mo.

*One patient living at present.

The microscopic diagnoses were as follows: adenocarcinoma, 82.7 per cent; squamous carcinoma, 4 per cent; carcinoma (cell type not specified), 13.3 per cent.

Cholelithiasis was found either at operation or at necropsy in fifty-five cases, or 73.3 per cent. In an additional 13 per cent where the disease was inoperable and no necropsy was performed, it is probable that stones were present. The remainder came to autopsy, but in only one case was it specifically stated that no stones were present, no reference to stones having been made in the others. In two cases, with a previous history of cholecystostomy, reformed stones were found and an associated carcinoma. This finding is similar to the experience of Finney and Johnson.[8]

DISCUSSION

With a mortality of 98.5 per cent in less than three years after operation this disease is virtually incurable. Early diagnosis is not an aid, for once the disease manifests itself by the production of symptoms it is already beyond the localized stage. This is well demonstrated in Table II where it is noted that even in eight cases fortuitously discovered by the pathologist, the average life following discovery was only thirty months, and eleven and one-half months if we exclude the only survivor. Radical and palliative procedures add only a few months to the survival period in those patients in whom the disease can be so treated. The over-all survival period following operation in all sixty-five cases is 4.4 months.

If in the light of our present knowledge we are unable to provide adequate active therapy for this disease, it seems imperative to apply preventive measures. Although the exact etiology of carcinoma of the gall bladder is as yet unknown, evidence that cholelithiasis is the most important predisposing factor is too strong to ignore. In the face of convincing statistics and experimental data, we must accept this as the most important precursor of carcinoma. Graham, in a review of statistical studies, found that the incidence of cholelithiasis in carcinoma of the gall bladder ranged from 69 to 100 per cent. Other significant series have shown 70 to 100 per cent,[7] 72.6 per cent,[2] and 80 per cent.[8] Our incidence is at least 73.3 per cent. All figures reported are many times the incidence of gallstones as reported in various necropsy statistics on patients dying from all other causes.[2, 8]

That foreign bodies in the gall bladder are capable of producing carcinoma and other less malignant forms of metaplasia has frequently been shown experimentally. Mohardt quoted the work of Kazama and Leitch, who inserted hard foreign bodies in the gall bladders of guinea pigs, resulting in carcinoma in twenty-six of ninety-eight, and eight of thirty-five animals, respectively. More recently Petrov and Krotkina[6] found five cases of carcinoma resulting from the introduction of foreign bodies into the gall bladders of fifty-one animals. They emphasized that even in these short-lived animals, it took a period of one to two years to obtain these carcinomatous changes.

That the stones are not a result of carcinoma is pointed out by Graham, who stated that in series of metastatic carcinoma of the gall bladder, the incidence of stones is only 8 per cent, well within the usual range of gallstone incidence in all patients. Papillomas of the gall bladder have been suggested as a predisposing factor; Mohardt quoted a series of 500 cases of papilloma of the gall bladder, without a single association of carcinoma in that organ.

If, then, cholelithiasis seems responsible for this malignant transformation, are we justified in performing cholecystectomies on patients with asymptomatic gall stones, as well as on patients with symptoms, because of the possibility of carcinomatous transition? The incidence of carcinoma in calculus gall bladders is variously reported as 3,[2] 5,[8] and 5 to 14[5] per cent. The mortality following cholecystectomy at various institutions compares more favorably. Mortality of 1.6 per cent following cholecystectomy for all patients, including those with

jaundice, was reported by the Mayo Clinic.[9] Graham gave a figure of 1.5 per cent. In 1946, at the Mount Sinai Hospital there were 346 cholecystectomies on patients with all the various complications of stones, resulting in the death of two patients, or an over-all mortality of 0.6 per cent. It follows that cholecystectomy is indicated for calculus gall bladder on the grounds that the operative mortality is less than the mortality due to malignant transformation. This, in addition to the other frequently fatal complications of cholelithiasis, constitutes an obligation to remove all calculus gall bladders, in the absence of surgical contraindications. Although there may be such an entity as "silent" gallstones, we have no method of foretelling which will remain as such.

SUMMARY

A report is made of seventy-five patients with carcinoma of the gall bladder, sixty-five of whom were operated upon, with only one survivor (thirteen years) longer than three years. When this disease is diagnosed clinically it is virtually incurable; consequently its occurrence must be prevented. Overwhelming evidence points to gall stones as a predisposing factor in carcinoma of the gall bladder. Since the death incidence of malignant transformation in calculus gall bladders far exceeds the prevailing operative mortality of cholecystectomy, it is indicated to remove even asymptomatic calculus gall bladders on these grounds alone.

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HEMORRHAGIC INFARCTION OF THE GREATER OMENTUM SIMULATING ACUTE APPENDICITIS

A Report of Two Cases

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FROM time to time reports have appeared in the literature of lesions of the greater omentum, variously classified as primary acute epiploitis,[1] idiopathic segmental infarction,[2] or as infarcts due to primary torsion of a segment of the greater omentum.[3] In all of these there is a common underlying pathology characterized by vascular congestion, hemorrhagic extravasation, usually a thrombus formation, and an inflammatory response of varying degree. Evidence of torsion of the involved portion of the omentum is a relatively frequent but not invariable finding. The clinical and laboratory picture is related most closely to that found in acute appendicitis, and the diagnosis can be made only at surgery.

The etiologic factors have been well discussed by Eliason and Johnson, who reported thirteen cases and reviewed the theoretical possibilities in the causation of this unusual lesion. The opinion of these writers is that the lesion is vascular in nature, resulting either from occlusion of the blood supply due to torsion, or from primary thrombosis or embolism of the omental vessels. A third possibility, bacterial invasion of the omentum by way of the blood stream, is mentioned but is not to be confused with the type of case under discussion.

That the lesion is a primary one of the omentum is shown by the fact that only in extremely few of these cases were there evidences of other systemic or vascular lesions that might cause these changes. In one of the cases reported by Pines and Rabinovitch, for example, there was demonstrated a pre-existing Buerger's disease; Hines[4] reported a case with primary infarction due to thrombosis of the superior mesenteric and splenic veins; and in Berger's case there was cardiac decompensation with arteriosclerosis, hypertension, and syphilis. A case of Totten's[6] presented a definite although compensated aortic insufficiency incident to a pre-existing rheumatic fever.

CASE REPORT

Case 1.—L. S., a 52-year-old white man, was admitted to the hospital with a complaint of pain in the right lower quadrant of seventy-two hours' duration. The pain was described as being dull and intermittent in character, the patient being able to sleep during the remissions. There was no nausea or vomiting, and no bowel changes were noticed. Past medical history was not contributory. The patient had been trying unsuccessfully to lose weight for several weeks.

Physical examination revealed an obese man lying in bed in no great distress. There was a definite tenderness and guarding to deep palpation over the right lower quadrant, but

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rebound and psoas tests were negative. In addition there was a harsh blowing systolic murmur heard maximally at the cardiac apex, which was transmitted up the left sternal border to the neck vessels. Temperature on admission was 98.6° F., pulse was 108, respiration was 18, and blood pressure was 160/90. Electrocardiogram showed depression of S-T_2 and S-T_3 with flattening of T_1 and T_2, indicating suggestive evidence of myocardial damage. The urine examination was negative and the blood showed a total white cell count of 8000, with 78 per cent polymorphonuclear cells and 22 per cent lymphocytes. Kline exclusion test was negative. The persistence of the right lower quadrant pain and discomfort for seventy-two hours with moderate guarding and tenderness over McBurney's point despite a fairly normal blood count appeared to justify a diagnosis of acute appendicitis, and operation was recommended.

Through a McBurney approach and under general anesthesia the abdomen was opened. Exposure was extremely difficult because of the large amount of fat present in the body wall and in the omentum. The appendix was exposed and was disappointingly free of gross evidence of inflammation. It was removed in the routine manner and further exploration of the abdomen was undertaken.

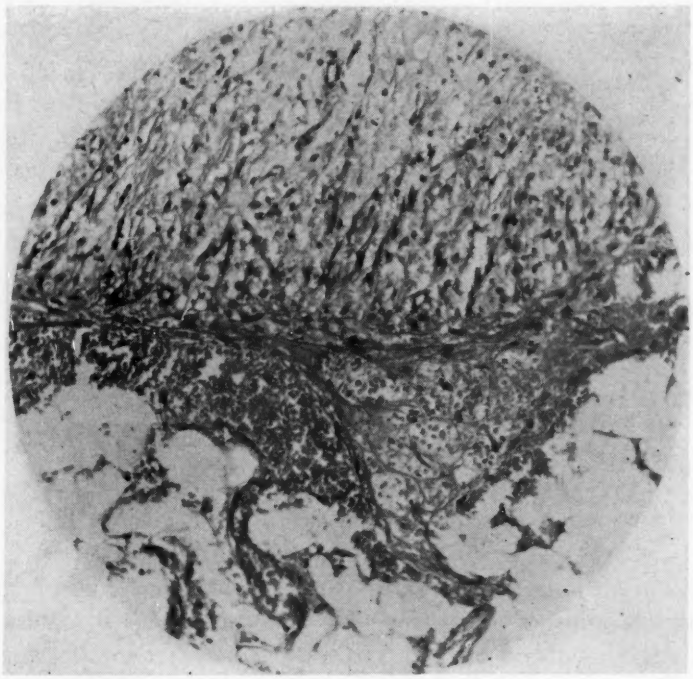

Fig. 1.—Photomicrograph of tissue taken from patient (Case 1). Note the dissolution of the fat and proliferation of fibrous tissue.

Lying immediately superior to McBurney's point and just beneath although not adherent to the parietal peritoneum was a reddish-black segment of the distal greater omentum, measuring approximately 3 by 3 cm. It resembled grossly an area of hemorrhagic necrosis but was unlike any pathologic entity previously seen by the surgeon. It was decided to remove this area of the omentum for histopathologic examination. The remainder of the abdominal exploration being negative, the abdomen was closed.

Microscopically this portion of omentum showed a diffuse acute hemorrhagic process with blood present under the peritoneal surface and extending along the septal fibrous bands. Associated with the red blood cells were groups of polymorphonuclear leucocytes, some of which were undergoing dissolution. In a few places the fat cells were considerably altered with a tendency toward granule formation in the cytoplasm. The septal fibrous bands were

somewhat masked by the acute hemorrhagic process and were considerably thickened by the proliferation of connective tissue cells and by edema, in addition to the leucocytes. One sharply demarcated zone showed an avascular organization with young fibroblasts replacing fat cells. No hemosiderin or other evidence of old hemorrhage was present. Throughout the tissue the blood vessels were dilated and congested but there was no evidence of specific vascular disease (see Fig. 1).

CASE 2.—W. S., a 49-year-old white man, entered the hospital with a complaint of abdominal pain for the preceding twenty-four hours. The onset was fairly sudden and the progress of the pain was constant until, at the time of admission, he was in acute distress. The pain was diffuse at first over the entire right side of the abdomen and flank, but localized over the right lower quadrant two or three hours before entry. The pain, although constant, was characterized by spasmodic increases in intensity, at which times the patient was obliged to flex the thighs on the abdomen. There was no nausea or vomiting. Past medical history revealed a "chronic appendicitis" ten years previously, the recurrent attacks of which were marked by a dull pain in the right lower quadrant which soon subsided on application of an ice bag.

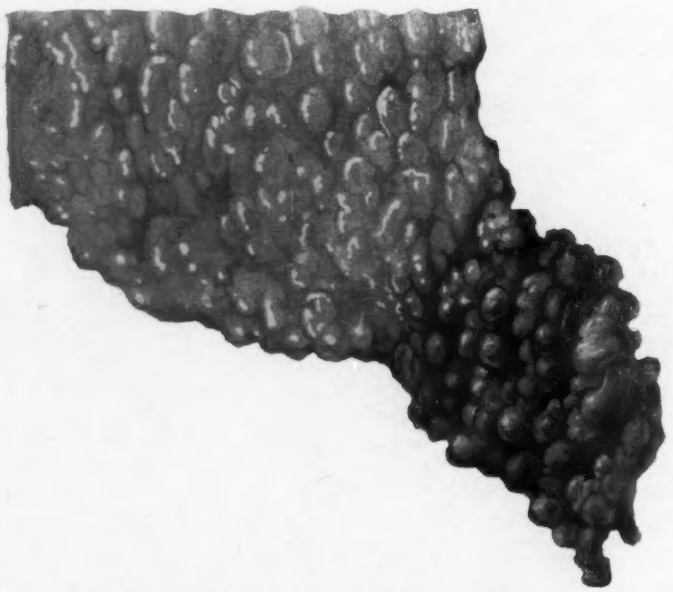

Fig. 2.—Artist's representation of specimen taken in Case 2. Note the sharp line of demarcation between normal and diseased tissue.

Physical examination revealed an obese individual and was negative except for diffuse tenderness and guarding, most marked over the right lower quadrant, with rebound tenderness and skin hyperesthesia elicited in this region. The abdomen was moderately distended and peristalsis was minimal. Temperature on admission was 100.4° F., pulse was 110, and respirations 25. The urine was negative; the blood showed a total white cell count of 5300 with 64 per cent poymorphonuclear leucocytes and 36 per cent lymphocytes. Kline exclusion test was negative. A diagnosis of acute appendicitis was made

Under spinal anesthesia the appendix was approached through a McBurney incision. Lying immediately beneath the parietal peritoneum and delivering into the operative field as this was opened was an irregularly ovoid, hemorrhagic mass measuring about 2.5 by 3 cm. (see Fig. 2). This was seen to be an appendage of the greater omentum, from the normal tissue of which it was sharply demarcated. This hemorrhagic tissue, as well as a moderately fibrotic appendix, was excised; the abdomen was closed in layers and the postoperative course was uneventful.

Microscopic examination of the tissue showed an acute diffuse interstitial hemorrhagic process with small groups of red blood cells arranged in clusters or cords between isolated fat cells. There was a superficial focal accumulation of leucocytes and fibrin among the red cells along the peritoneal aspect of the fat. All the vessels were dilated and congested, but showed no specific alteration.

Both cases simulated acute appendicitis clinically although admittedly the white blood count was low, remaining well below the average of 12,000 found in the six cases reported by Pines and Rabinovitch. In both cases tenderness and rigidity were present, nausea and vomiting were absent, fever was slight. These observations are not out of harmony with the results of the aforementioned workers or with those of Eliason and Johnson. At operation the appendix was not involved in either case, but the clinical picture is explained by the pathologic findings in the greater omentum; this also coincides with the experience of previous writers.

It can be seen from a comparison of the two cases that the pathologic process in the first instance has advanced far beyond that noted in the second, and this is borne out by the clinical history. Evidence of advanced necrosis, as described by Pines and Rabinovitch, is lacking but we believe this to be due to the recent occurrence of the hemorrhage. In neither of the cases was thrombosis a causative factor or a consequence of the production of the lesion. In neither case was there torsion of the involved omental segment, nor was there evidence that this had occurred earlier with subsequent restitution of the twisted segment.

It is interesting to observe that both patients were of a hypersthenic body habitus, and both had abdominal walls and greater omenta that were heavily laden with fat.

The absence in both cases of evidence of torsion and the inability to point out evidence of specific vascular lesions immediately raise the question of the etiologic factors involved. The presence of presumptive evidence of cardiac damage in Case 1, as shown by the electrocardiogram, could not be explained on a basis other than the mild hypertension, and the patient was well compensated. Vascular sclerosis, as indicated by eye-ground changes, was minimal; arterial emboli were unlikely on the basis of the consistently regular cardiac rhythm, by the absence of other evidence of embolic phenomena, and by the nature of the pathologic process in the infarcted area itself.

The lesion, therefore, must be regarded as a localized process. Experiments performed on animals by Pines and Rabinovitch indicate that a sudden pull along the axis of a vessel can lead to damage of the intima and production of a thrombus closely related in type to those observed in their cases. However, it is pointed out that in neither of our cases was there evidence of thrombus formation, any infarct formation being due, apparently, to the primary hemorrhage.

Totten hypothecated a state where the thin-walled omental vessels, engorged following a meal, are subjected to an unusual force incident to straining, sneezing, and so forth, which is sufficient to rupture a vessel and cause hemorrhage with the sequence of pathologic changes just enumerated. Indeed, our

two cases resemble most closely (from a pathologic standpoint) the second of the two cases reported by him. Neither of our patients, however, offered any history of previous violence or straining, such as was recorded in the case of Totten.

It is believed by us that an extremely fatty omentum, as was seen in both our cases by increasing the gravitational pull acting on the omental vessels may act in conjunction with the factors mentioned to cause rupture of a vessel.

CONCLUSIONS AND SUMMARY

1. Two cases of primary hemorrhagic infarction of a segment of the greater omentum are presented, together with a discussion of the pathologic changes found in each case.

2. Possible factors in the causation of this lesion are reviewed.

3. The inability to differentiate this condition from the clinical manifestations of acute appendicitis or other intra-abdominal conditions is emphasized.

4. It is believed by us that the correct diagnosis may be suspected if consideration is given to the fact that the syndrome occurs more commonly in hypersthenic, obese individuals.

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A COMPARATIVE STUDY OF THE ACTION OF DEMEROL AND OPIUM ALKALOIDS IN RELATION TO BILIARY SPASM

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THE chemical structure of demerol* was originally designed to resemble that of atropine and papaverine combined.[1] This was done in order to combine the neurotropic action of the atropine and the musculotropic action of papaverine in the hope that a double acting smooth muscle relaxing drug would result. Animal experimentation soon demonstrated its unexpected analgesic properties as well. Further investigation revealed that the opium alkaloids were shown to be related chemically to demerol.[2] The central analgesic action of demerol has since been well established, and it is because of this property that it finds its greatest use today. It has found a place in the clinician's armamentarium where analgesia approaching that of morphine is desired without the undesirable side effects of the latter, such as respiratory depression, constipating action, urinary retention, and depression of cough reflexes. This is most notably true in obstetrics, in pre- and postoperative care, particularly in the face of pulmonary pathology, and in poor-risk or elderly patients. The original findings of its nonaddicting properties have not been substantiated. Tolerance, physical dependence, and habituation have been shown to develop with its prolonged use.[3-10]

It is our purpose in this paper to attempt an evaluation of the spasmolytic properties of demerol with particular reference to the biliary tract and to the second portion of the duodenum.

REVIEW OF THE LITERATURE

The spasmolytic action of this drug was first demonstrated in animals by Eisleb and Schaumann.[1, 2] Strips of large intestine of the guinea pig which had been made spastic by such substances as acetylcholine, barium chloride, or histamine were promptly relaxed by demerol. It was noted, however, that the effect was variable in different sections of the intestine. Furthermore, the drug's action on quiescent segments was not tested, or at least not reported. Gruber, Hart, and Gruber[11] after extensive investigation on the pharmacologic action of demerol in this country confirmed the antagonism to spasm from histamine, barium chloride, or acetylcholine. They demonstrated, however, that in the intact intestine in dogs, as well as in isolated duodenal and ileal strips of cats and rabbits, the drug regularly increased tonus and peristalsis and decreased the organ's volume at the same time. The latter action they attributed also to smooth muscle spasm. Furthermore, they showed that demerol has no relaxing

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*1-Methyl 4-phenyl piperidine 4-carboxylic acid ethyl ester hydrochloride, also known in various countries and at various times as Eucolat, Dolantol, Dolantin, Pethidine, and meperidine hydrochloride.

effect on bowel made spastic with morphine. The former finding was amply confirmed by Yonkman and his co-workers,[12] but they claimed that demerol relaxed the spasm produced by morphine in the intestine. This was also confirmed by Dryer.[13] Barlow[14] found antagonism to histamine, barium chloride, and, in addition, to pilocarpine and physostigmine. He, however, found a spasmolytic effect on quiescent ileal strips of rabbits. Observations made in Great Britain by Duguid and Heathcote[15] showed the drug to be one-half as effective as papaverine in reducing amplitude of contraction of an isolated gut segment. On the acetylcholine activated intestine, they found atropine to have 500 times as much relaxing quality as demerol. As the drug was also found to slow respirations, depress the blood pressure, and decrease the amplitude of heart beat, they concluded that demerol is a depressant to all forms of muscular tissue. Batterman and Himmelsbach[8] summarized these results of animal investigation and pointed to the variable and conflicting results of the effect of demerol not only on smooth muscle of the intestinal tract, but on that of other organs as well, such as the uterus.

In man there have been but few studies to substantiate the expected relaxing effect on the intestinal and urinary tracts, which is the basis of its use in biliary, intestinal, and renal colic. Decrease in tonus and decrease in amplitude of contraction with use of demerol on the human ureter has been demonstrated in one study.[16] Investigations of the gastrointestinal tract have been confined to balloon recordings either by intubation or through incidental fistulas. Batterman[17] reported an antispasmodic effect in 84 per cent of twenty-seven human subjects studied. His records, however, showed a decrease in motility rather than in tone. This is in contrast to his clinical experience in which he found the drug nonconstipating. The decrease in motility was most marked in the stomach and pylorus and slight in the ileum and colon. There was no report of studies on the duodenum in his paper. His results have been confirmed by other investigators.[13, 14] Yonkman[18] found that the drug did not decrease tone but decreased large intestinal contractions. He could not, with demerol, prevent the tonic action of morphine. No studies on the biliary tract have been reported.

Clinically, the use of demerol in pain due to spasm of smooth muscle of intestinal, biliary, and renal colic has been advocated on the basis of the experimental results described, or with an eye to its chemical structure and intended effects. Frequent final recourse to morphine for this type of pain is admitted by some authors.[19] Others recommend demerol as the drug of choice for this type of pain and mention excellent and even dramatic results with its use, but no detailed statistics are given.[8, 17, 20, 21] Noth and his co-workers[7] reported relief in only three of five cases of biliary colic.

We have undertaken a study of demerol and its pharmacodynamic action on the biliary tract and second portion of the duodenum for several reasons: (1) We have not been able to confirm the clinical results of others of dramatic relief with demerol in biliary colic; (2) it has been our impression for some time that demerol has no beneficial spasmolytic action on the biliary tract and that the relief, if any, is due to its central analgesic action; and (3) previous experi-

mental work has appeared inconclusive to us. Further, the spasmolytic action of demerol is attributed to its atropine-like and papaverine-like structure. Both these drugs have been shown by Butsch, Walters, and one of us (J. M. M.)[22] to be ineffective in biliary spasm.

METHOD

In a previous publication Butsch, Walters, and one of us (J. M. M.)[23] has described a method of measuring pressure in the common bile duct of man. Measurement of pressure changes was found to be an excellent method of studying changes in the tone of the musculature of the second portion of the duodenum.[24] It was found that spasm of the muscle of the second portion of the duodenum occluded the lower end of the common bile duct with a sphincter-like action. It was concluded that while the sphincter of Oddi may exist as a separate entity its function is physiologically synchronous with that of musculature of the second portion of the duodenal wall. For this reason the term, duodenal spasm, refers to spasm of the sphincter mechanism of the biliary tract (sphincter of Oddi). It was found that spasm of the second portion of the duodenum was produced by codeine, dilaudid, pantopon, and morphine. This spasm produced enough back pressure in the biliary tract, in some cases, to produce an attack of biliary colic. The nitrite drugs, aminophylline and epinephrine, were found to relax this spasm, while papaverine and atropine were without effect. This same method of pressure studies has been found useful in determining the optimum time for removal of the T tube.[25-27]

Pressure studies made include (1) resting intrabiliary pressure, (2) perfusion pain level, and (3) pressure changes resulting from the action of various drugs. Ten postoperative patients with T-tube biliary drainage were studied. Pressure studies were made under various conditions in order to compare the action of demerol with opium alkaloids on the sphincter mechanism at the lower end of the common bile duct. These patients had been subjected to an exploration of the common bile duct for such varied pathology as cholelithiasis with "gravel," dilation of the common duct, common duct stone, history of jaundice, pancreatic fibrosis, and chronic pancreatitis. It is to be noted that in five of these ten patients common duct stones were found at operation.

The pressure apparatus used was similar to the one previously described.[23] Briefly it consisted of a water manometer with a thinly blown glass bulb as float. An aluminum extension and writing arm was attached to this float and arranged to record the pressure in millimeters of water on a slowly revolving smoked drum. The manometer was connected to the T tube emerging from the abdominal wall and to a bottle of sterile physiologic saline solution used to fill the system. A marker was used on all kymographic recordings. The base line was adjusted to correspond to the level of the xiphoid process of the patient, which was considered the zero level. The patient's resting intrabiliary pressure was recorded on the drum for about one-half hour before any drug was given. Three deep inhalations of amyl nitrite were then given to determine the presence or absence of initial spasm.[25] Following this the various drugs under investigation were administered. This procedure was carried out on the patient for the first

time two weeks postoperatively and repeated until the biliary dynamics returned to normal.[26, 27] Weekly cholangiograms (using diodrast) were made in order to detect any missing stone and to follow the progress of any obstructing lesion such as ampulla edema, spasm, or pancreatitis, and to study the effect of the various drugs.

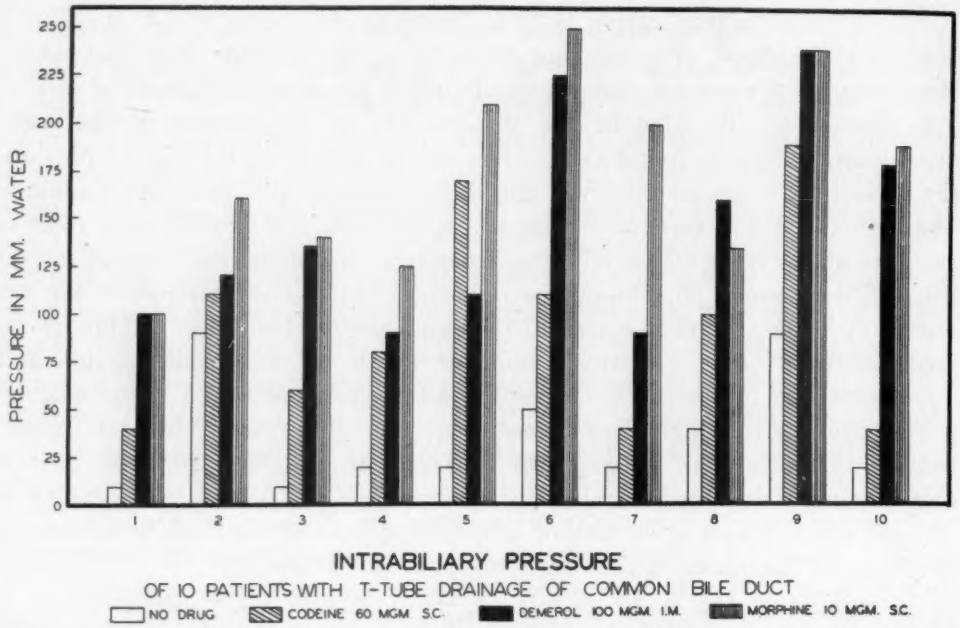

Fig. 1.—Comparison of the spasmogenic action of codeine, demerol, and morphine on the sphincter mechanism of the common bile duct as measured by changes in the intrabiliary pressure in man.

RESULTS

Demerol was found to produce spasm of the sphincter mechanism of the common bile duct (duodenal wall). The resultant spasm, as evidenced by a rise in intrabiliary pressure, was found to begin within five minutes after administration of 100 mg. of the drug intramuscularly. Pressure then rose

TABLE I

PATIENT	RESTING INTRA-BILIARY PRESSURE	PEAK READING WITH		
		CODEINE (60 MG. S.C.)	DEMEROL (100 MG. I.M.)	MORPHINE (10 MG. S.C.)
1	10*	40*	100*	100*
2	90	110	120	160
3	10	60	135	140
4	20	80	90	125
5	20	170	110	210
6	50	110	225	250
7	20	40	90	200
8	40	100	160	135
9	90	190	240	240
10	20	40	170	190
Mean value		94	144	175

Note: These figures represent peak values in millimeters of water taken from kymographic tracings. They were taken from numerous records selected for comparison.

quickly to a peak usually reached at the end of twenty minutes. Some spastic effect was found to last for as long as 1½ to 2 hours (Figs. 2 and 3). The intensity of the spasm was intermediate between that of codeine and that produced

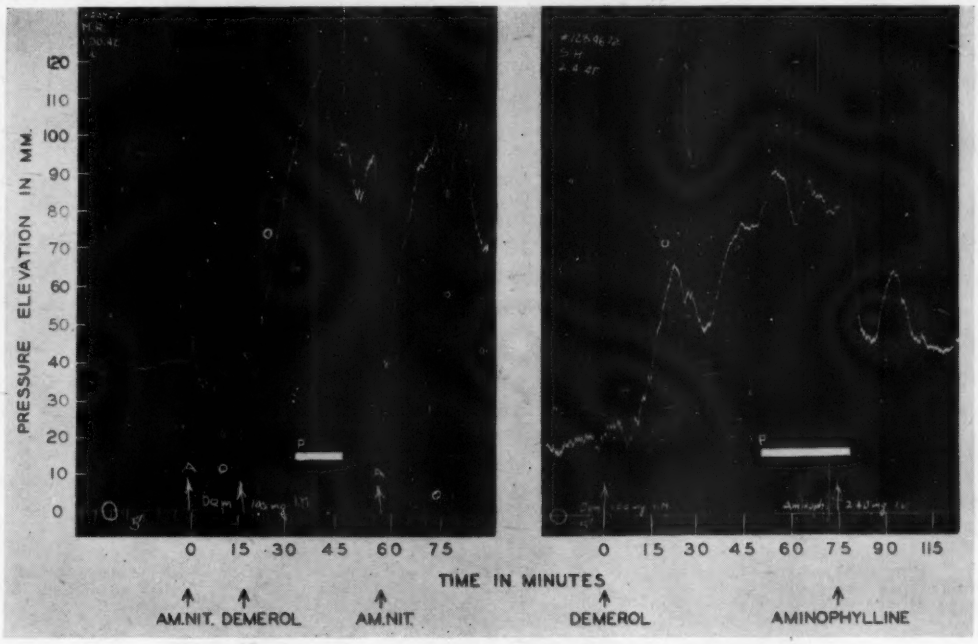

Fig. 2.—Kymograph tracings showing pressure changes associated with two instances of biliary colic produced in different patients by the administration of 100 mg. of demerol intramuscularly. Relief in the first case was obtained with inhalations of amyl nitrite and in the second with 240 mg. of theophylline with ethylenediamine (aminophylline) given intravenously. *P*, pain. The base line or zero level in this and succeeding illustrations is at the level of the xiphoid with the patient in the prone position.

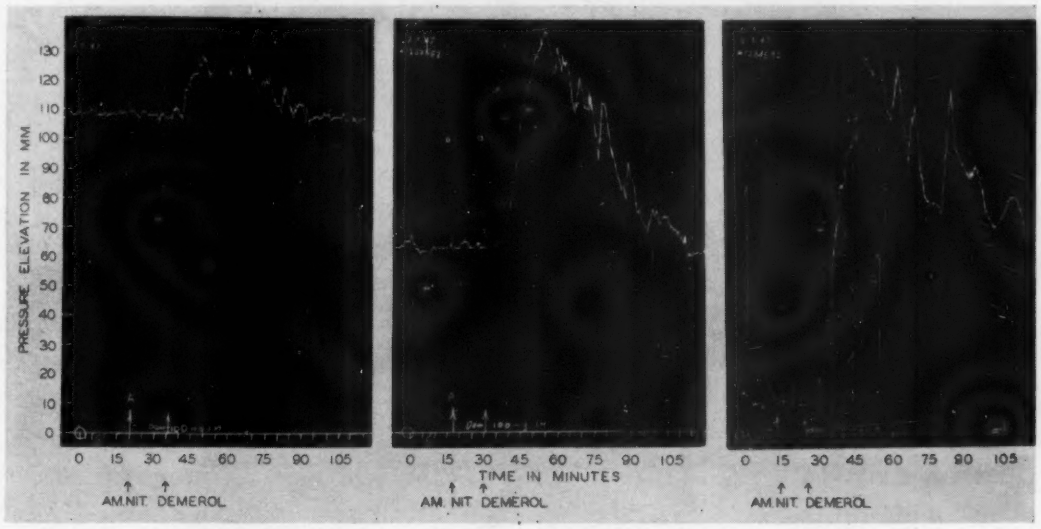

Fig. 3.—Three tracings taken two, three, and four weeks postoperatively in the same patient. No pressure reduction to amyl nitrite initially indicates edema rather than spasm as the cause of the elevated resting pressure. Here edema decreased gradually postoperatively as noted in each succeeding graph. Demerol produced the same peak elevation from a variable resting level.

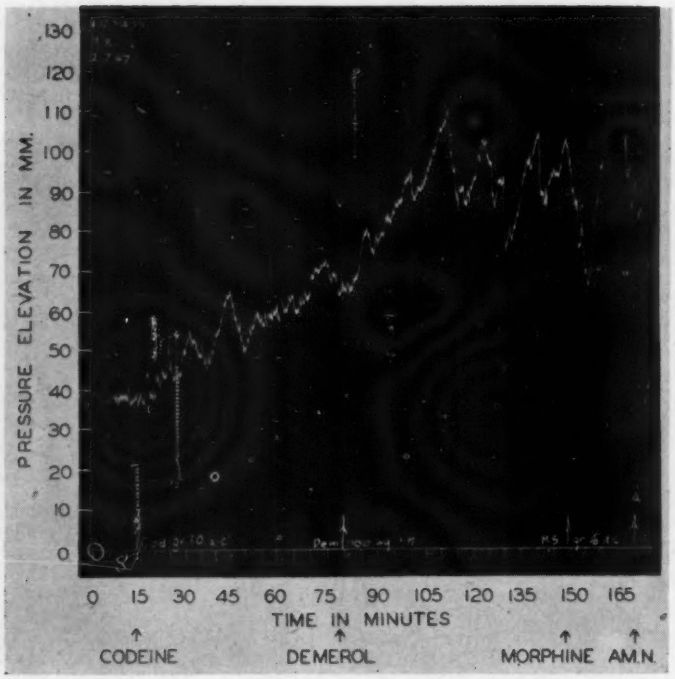

Fig. 4.—Codeine, demerol, and morphine administered consecutively in the same patient, illustrating the spasmogenic properties of these drugs in the order named. Relief of spasm by amyl nitrite occurred at the end.

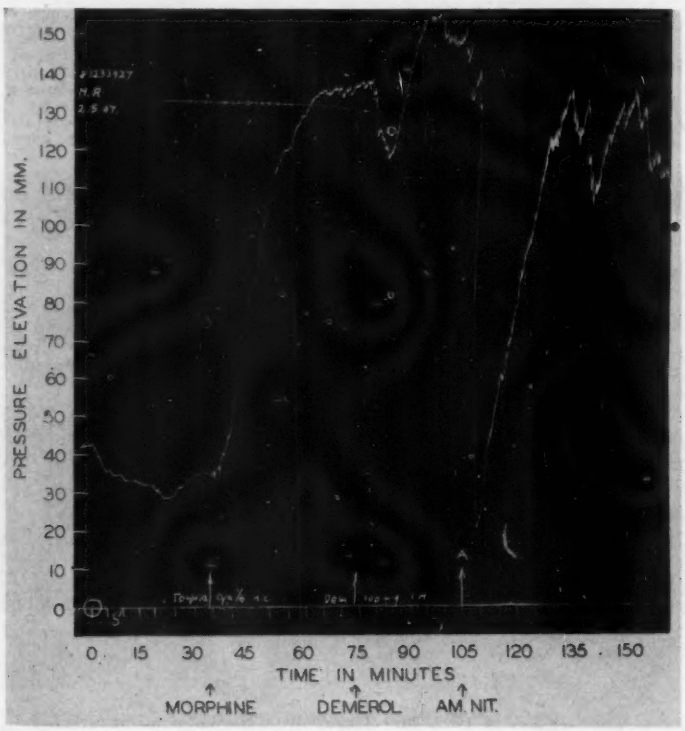

Fig. 5.—Demonstration of the inability of demerol to relieve morphine-induced spasm. In this particular patient demerol produced more spasm than did morphine. Relief was obtained with amyl nitrite.

by morphine (Figs. 1 and 4).[22] Generally the intensity of spasm approached that of morphine. Table I illustrates this point. The average intrabiliary pressure elevations, above normal, recorded in millimeters of water for the three drugs studied, are as follows: codeine, 94; demerol, 144; morphine, 175. In one instance demerol was even more spasmogenic than morphine (Figs. 1 and 5).

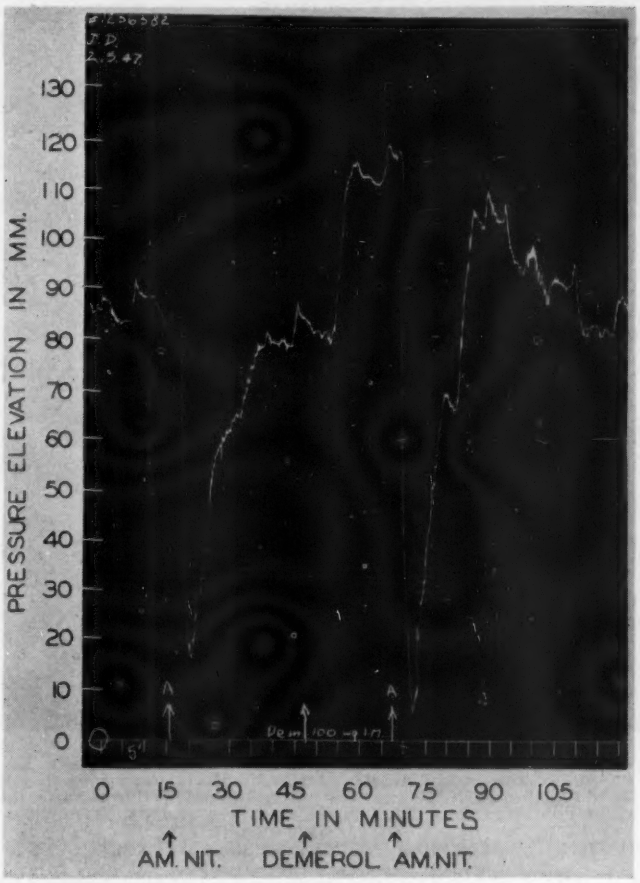

Fig. 6.—Example of natural duodenal spasm (not drug) relieved by amyl nitrite and increased rather than relieved by demerol.

When the drugs were given consecutively to the same patient, the spasmogenic effect of the second drug was found to be superimposed on that of the first (Figs. 4 and 5). In some cases pressure elevation was present from edema of the ampulla of Vater. Such a pressure elevation naturally does not decrease with amyl nitrite (Fig. 3). The ampulla edema gradually decreased with continued T-tube biliary drainage. Fig. 3 shows that the degree of edema does not effect the pressure elevation due to demerol. As the edema and resting pressure level decreased with T-tube drainage, the height of pressure elevation from demerol remained the same. This means that a patient with biliary colic from back pressure as a result of obstruction due to a stone or edema is not, so far as biliary dynamics are concerned, made worse by demerol. In other words, if a

patient with biliary colic is given amyl nitrite and no relief is obtained, one may then assume that the back pressure is due to some cause other than spasm and may administer demerol or an opiate derivative for relief of pain and know that the biliary dynamics will not be further disturbed.

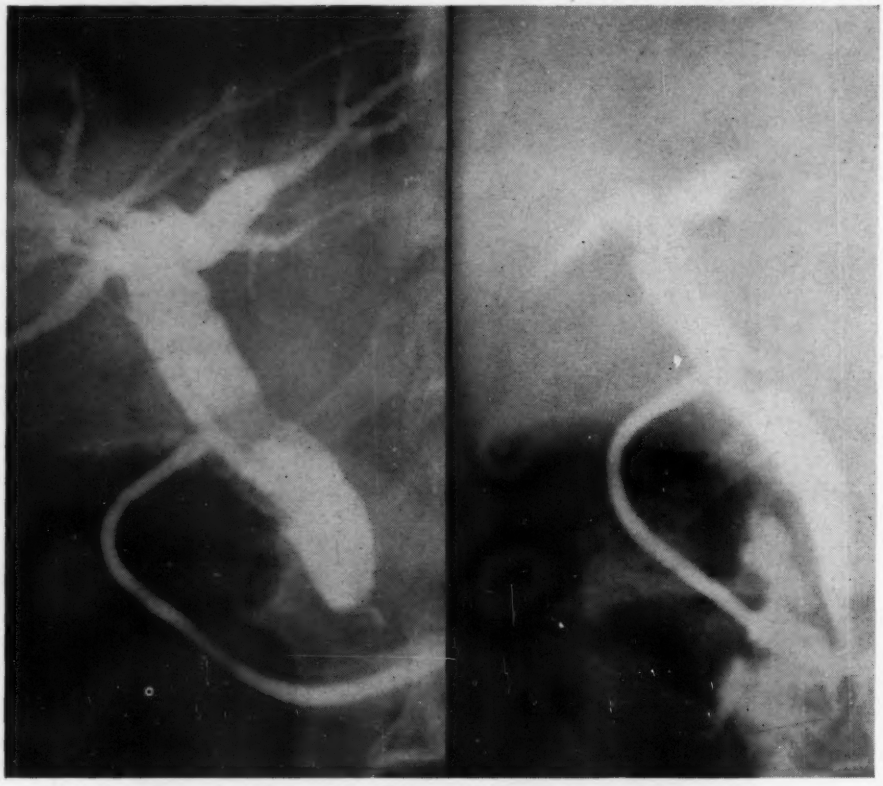

Fig. 7.—*A*, Cholangiograms of the same patient as illustrated in Fig. 6. There is marked spasm at the lower end of the common bile duct with dilatation above (hydrohepatosis). No diodrast reached the duodenum. *B*, The second plate was taken one minute following inhalation of amyl nitrite. The sphincter has relaxed and contrast media is flowing freely in to the duodenum.

Demerol, hence, was found regularly to increase the spasm already produced by a previous dose of codeine (Fig. 4). Demerol given after morphine was found not to relieve the spasm (Fig. 5). The spasm produced by demerol was regularly but only briefly relieved by either inhalations of amyl nitrite or intravenous injection of theophylline with ethylenediamine (aminophylline) (Figs. 2, 4, 5, 6).

Two patients experienced a typical attack of biliary colic with nausea and cramping right upper quadrant pain following administration of demerol about two weeks postoperatively. These attacks were relieved by amyl nitrite inhalation in one case and by aminophylline intravenously in the other (Fig. 2).

In addition to its failure to relax morphine spasm, demerol was found to be ineffective against natural spasm. On the contrary, it further increases this spasm (Fig. 6). Cholangiograms taken five to thirty minutes after intramuscular administration of 100 mg. of demerol repeatedly demonstrated these

spasmogenic qualities of the drug. Relief with amyl nitrite was well visualized (Fig. 5). The pain threshold as determined by perfusion pain levels was regularly raised by demerol, its action in this respect being intermediate between that of morphine and that of codeine. In other words, it does not produce as much spasm as morphine but it does not relieve pain quite as well, either. The only side effects noted with administration of demerol intramuscularly were perspiration and slight dizziness. These were found in 100 per cent of the subjects studied.

CONCLUSION

Demerol is not the answer to the quest for an ideal spasmolytic analgesic agent for use in biliary colic and postoperative biliary pain. If biliary colic is due to spasm, nitroglycerin, gr. $\frac{1}{250}$,[25] in doses of one to two granules under the tongue will relieve the attack. This should be tried first in such instances. If the pain is due to biliary obstruction other than spasm, no relief will be afforded by the nitrites and there will not be harmful aggravation of the local pathology by the administration of spasmogenic analgesic. In such an instance, morphine or demerol may be given for the relief of this severe type of pain. For postoperative use demerol is not contraindicated if relief of pain cannot be obtained by the nitrite drugs. It does not elevate the pressure above the secretion pressure of the liver (300 ml. as had been shown in the case of morphine[23] and, therefore, will not tend to produce jaundice. If following cholecystectomy, biliary fistula is feared or is present, demerol should be used with as much caution as any drug which raises intrabiliary pressure.

SUMMARY

1. Contrary to common belief, demerol has been shown to be a spasmogenic rather than a spasmolytic agent on the sphincter mechanism at the lower end of the common bile duct (Oddi) in ten unselected patients with T-tube drainage of the common duct.

2. The spasm produced was intermediate in intensity between that produced by codein and that of morphine; it was sufficiently severe in two cases to produce a typical attack of biliary colic.

3. Demerol was shown to increase rather than relieve natural spasm as well as spasm produced by codeine.

4. The effect produced by demerol was regularly but briefly relieved by amyl nitrite or theophylline with ethylenediamine (aminophylline).

5. Demerol should be used with as much caution as the opiates in post-cholecystectomy pain.

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SURGICAL MANAGEMENT OF ACQUIRED STRICTURE OF THE ESOPHAGUS WITH ESOPHAGOBRONCHIAL FISTULA AND BRONCHIECTASIS OF ENTIRE RIGHT LUNG

Report of Case

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BRONCHIECTASIS involving an entire lung does not occur frequently although it is not a rare condition. Strictures of the esophagus occurring as a result of ingestion of lye are, unfortunately, rather common. Acquired fistulas between the esophagus and the major bronchi or trachea are unusual. The occurrence of all three of these conditions in the same patient is certainly remarkable and presents formidable problems in regard to management. A report of such a case in which surgical management was successful appears warranted.

REPORT OF CASE

A white man, 25 years of age, registered at the Mayo Clinic on Aug. 24, 1946. At 2 years of age he had accidentally swallowed lye, and a stricture of the esophagus had developed. This had been treated with repeated esophageal dilatations and the patient had been able to take a fairly normal diet. At the age of 12 years he had pneumonia. The periodic esophageal dilatations were not performed during this illness and the esophagus became completely closed. Gastrostomy had been performed and all food and liquid had been given by this route for the thirteen years previous to admission to the clinic. Since the age of 2 years, when the patient had swallowed lye, he had been troubled with a mild, chronic cough productive of some mucopurulent material. After the patient had pneumonia at the age of 12 years, the cough became much worse. Three to four ounces (90 to 120 c.c.) of thick, purulent, blood-streaked sputum were raised daily. The patient had never been able to work. Clubbing of the fingers had developed after the pneumonia. Recently there had been rather severe hemoptysis. The patient had noted that if the feedings given through the gastric stoma were too thin or if they were too large he would cough up some of the food that had been administered. A diagnosis of bronchiectasis of the right lung and of a fistula between the right bronchus and esophagus had been made elsewhere.

On physical examination the patient was observed to be a tall, very thin, white man, weighing only 110 pounds (49.9 kilograms). There was no expansion of the right side of the chest, and the interspaces were narrowed. The right side of the chest was dull to percussion. Numerous coarse râles were heard on auscultation. There was marked clubbing of the fingers and toes. A gastric stoma, with tube in place, was noted in the left upper part of the abdomen. Physical examination was not remarkable otherwise.

The concentration of the hemoglobin was 14.2 Gm. per 100 c.c. of blood. The leucocyte count was 14,600 per cubic millimeter of blood with a normal distribution. The sedimentation rate was 32 mm. in one hour (Westergren method). Examinations of sputum for acid-fast bacilli gave negative results. Roentgenograms of the chest revealed an extensive suppurative process involving the entire right lung (Fig. 1). Bronchographic studies demonstrated diffuse saccular bronchiectasis of the right lung. There was a small area of cylindric bronchiectasis in the left base. An attempt was made to examine the upper part of the esophagus with

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radiopaque oil, but it was impossible for the patient to swallow enough oil to permit demonstration of anything. Apparently the upper part of the esophagus was completely closed. Barium was injected into the stomach through the gastric stoma, and the lower part of the esophagus was visualized satisfactorily. A fistula from the esophagus to the right bronchus was demonstrated.

Esophagoscopy was attempted. There was scarring at the level of the cricoid cartilage. About 3 cm. below the cricopharyngeus muscle the esophagus narrowed to a tiny slit and it was impossible to pass the esophagoscope beyond that point. Bronchoscopy was then performed. A large amount of pus was seen coming from the right lung. There was considerable inflammatory reaction in the right main bronchus. The fistulous communication with the esophagus could not be visualized. Subsequently esophagoscopy was again performed and attempts were made to pass filiform bougies through the strictured esophagus, but without success. A cystoscope was inserted into the stomach through the gastric stoma and attempts were made to pass ureteral catheters up the esophagus. Complete obstruction was met about 8 to 10 cm. above the cardia.

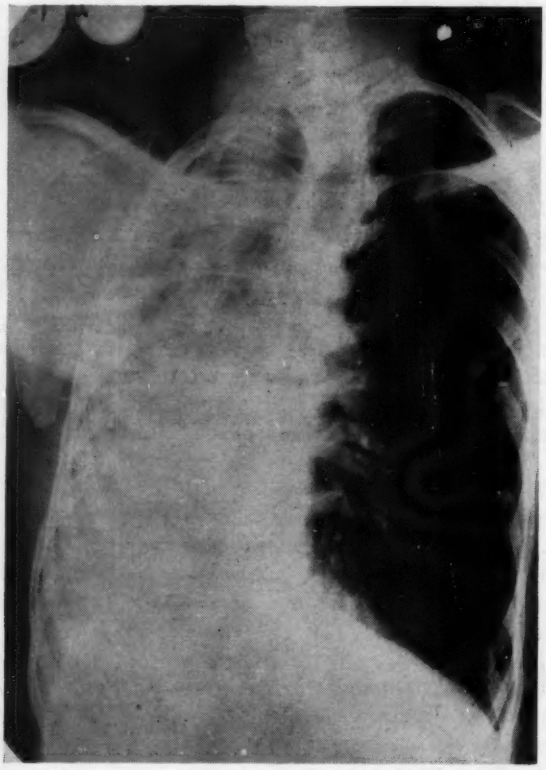

Fig. 1.—Extensive suppurative process throughout the entire right lung before operation.

All attempts to open the esophagus having failed, it was decided that the patient should undergo surgical exploration. After thorough preparation, including administration of penicillin, operation was performed on Oct. 10, 1946. A long, curved incision was made around the tip of the scapula on the right. The fifth rib was resected and the pleura opened. The lung was densely adherent to the parietal pleura over its entire surface and was mobilized with some difficulty. In some places it was necessary to carry out the dissection along an extrapleural line of cleavage. The hilar dissection was difficult, but each vessel was dissected out individually, doubly ligated, and sectioned. The posterior surface of the right main bronchus was densely adherent to the esophagus. The esophagus was so scarred that it was impossible

to establish definitely the location of the fistula. The bronchus was severed, the lung removed, and the stump of bronchus closed with a single row of interrupted silk sutures.

Below the level of the bronchus the esophagus appeared normal externally, but when its lumen was opened several short strictured portions were observed; a ureteral catheter could be passed into the stomach, however. A cystoscope was inserted through the gastric stoma, and the end of the ureteral catheter was picked up and brought out of the stomach through the gastric stoma. The esophagus above the bronchus was so scarred and fibrotic that it could hardly be recognized. A lumen was found finally and a ureteral catheter passed up toward the mouth. An esophagoscope was inserted, and this catheter was picked up and brought out through the mouth. A strong silk fishline suture was attached to the ureteral catheter and one end was drawn out through the mouth, the other out through the gastric stoma. Attempt was then made to repair the esophagus as well as possible, with pleura being used to cover the defects. The bronchial stump was also covered with pleura. The operation required about four and one-half hours. The patient was given 2,500 c.c. of blood during the operation.

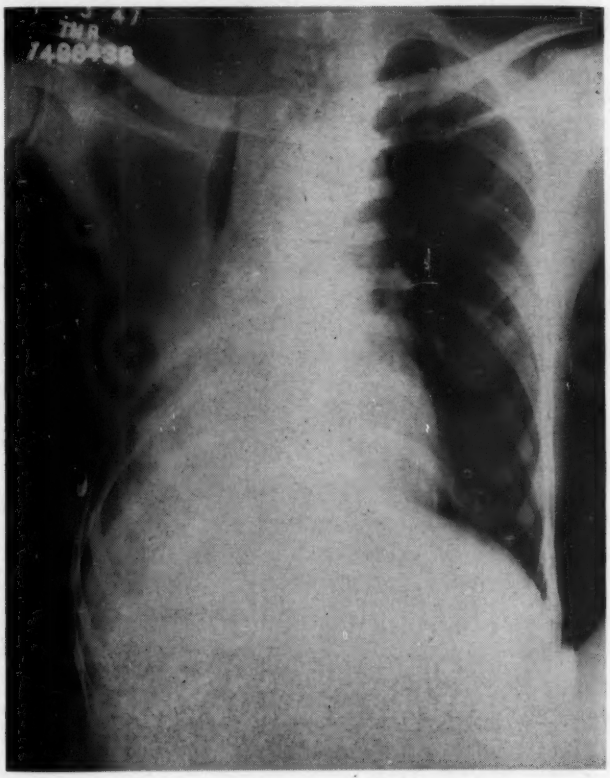

Fig. 2.—Appearance after pneumonectomy and thoracoplasty on the right.

Bronchoscopy was performed at the conclusion of the operation. A large amount of thick, mucopurulent material was aspirated from the trachea and the left bronchus. The patient was taken from the operating room in good condition. The immediate postoperative course was satisfactory, and the highest temperature was 102° F. On the seventh postoperative day evidence of considerable pneumothorax on the right developed. It was thought likely that the esophagus was leaking. There was no evidence of any leakage from the bronchial stump. It had been anticipated that such a leak might occur and cause empyema. On Oct. 19, 1946, with the patient under local anesthesia, the anterior end of the incision was opened and the pleural space evacuated of all its contents. The wound was left open to permit free drainage. One month later thoracoplasty was performed, the first seven ribs being removed (Fig. 2). Since permanent interruption of the phrenic nerve had been performed at the time

of pneumonectomy, thoracoplasty obliterated the remaining pleural space. On Nov. 26, 1946, progressive dilatation of the esophagus was instituted by passing dilators over the silk thread that had been left in place in the esophagus. The first dilatation was performed with a 22 French sound and subsequently larger sounds were used. The cavity caused by empyema became obliterated rapidly and was completely healed on Jan. 18, 1947. On dismissal of the patient the esophagus had been dilated up to the size of a 33 French sound and the patient was able to swallow all liquids and soft foods without difficulty (Fig. 3). He had no cough. He had gained weight and the general condition was excellent. Esophageal dilatations were continued by local physicians. The patient is now able to work for the first time in his life and is employed full time in a grocery store.

Fig. 3.—Appearance of the esophagus after closure of the fistula and progressive esophageal dilatations.

COMMENT

From the history in this case it seems likely that the bronchiectasis may have originated at the time the lye was accidentally swallowed. Possibly some of the lye was aspirated into the right bronchus, setting up an inflammatory process there, or some gastric contents may have gotten into the tracheobronchial tree during the vomiting that undoubtedly followed the swallowing of the lye. In a child 2 years old such an etiologic factor seems reasonable. There was no history of cough before the time lye was swallowed. We cannot be sure just

when the fistula between the esophagus and right main bronchus developed; it had not been noticed before the attack of pneumonia which occurred when the patient was 12 years of age, but it was apparent soon afterward. We believe that the development of the fistula may have caused the severe pneumonia that occurred in the bronchiectatic lung. The symptoms of bronchiectasis had been progressive since the development of the fistula and may have been due, in some part at least, to aspiration of food into the right lung when the feedings through the gastric stoma were too large or too liquid in character.

We had hoped that we might be able to get a string through the esophagus before attempting pneumonectomy, but this was impossible; if it had been possible the operation would have been somewhat easier than it was. The extent of the esophageal stricture and the involvement of the upper part of the esophagus almost to the cricoid cartilage precluded any attempt to excise the stricture and re-establish esophagogastric continuity by bringing the stomach high in the chest and anastomosing it to the upper part of the esophagus according to the method reported by Kay[1] and by Sweet.[2] This would not have been feasible in any event because the right lung was completely destroyed in so far as respiratory function was concerned, and an operation through the left side of the chest could not be considered. After careful consideration of the whole problem, we felt that our only chance of helping this patient lay in performing pneumonectomy on the right side and in attempting, at the same time, to repair the esophageal fistula and to reopen the strictured esophagus, if possible. The magnitude of such an operation is obvious. As we anticipated, it was a difficult procedure throughout. We were gratified that we were able to reopen the strictured esophagus and get a silk thread through from the mouth to the gastric stoma so that dilatation of the esophagus could be performed subsequently.

A strictured esophagus that can be re-utilized as a tube to convey food from the mouth to the stomach will function far better than an artificially made esophagus or an esophagogastric stoma made after esophageal resection. Uncomplicated cicatricial strictures of the esophagus can almost always be managed by dilating the stricture over a previously swallowed twisted silk thread. If anything will pass the strictured region, thread can be swallowed. If 15 feet (4.5 meters) of thread are swallowed at the rate of 1 foot (30 cm.) an hour the distal end will become anchored in the loops of the jejunum. Graduated Plummer dilating sounds can then be used to dilate the stricture, the thread serving as a guide. If there has been complete closure of the esophagus, a gastric stoma will be necessary. At the time of operation a thread can usually be passed into the stomach by attaching it to a flexible bougie and carefully introducing this instrument past the strictured region into the stomach, where the surgeon can disengage the thread and carry it out through the gastric stoma. At other times it is possible to thread a small ureteral catheter into the cardia in a retrograde fashion as has been described herein. Once a thread has passed through the lumen of the esophagus and has become engaged, repeated dilations are usually easy. The risk of these procedures is small and the results are excellent. One concern of ours in this particular case was whether or not a repaired esophagus

would withstand the trauma of esophageal dilation. This concern proved to be unnecessary since the patient stood the dilations very well.

We were not surprised that empyema developed. It would have been remarkable if it had not occurred. We had planned, in any event, to perform thoracoplasty, since we felt this would result in considerable support for the esophagus so that rupture of the esophagus during dilatations might be prevented.

The postoperative result has been excellent. The patient is able to swallow most foods. The gastric stoma has not been closed, but all feedings are taken in a normal manner. The patient is completely relieved of cough. For the first time in his life he is able to work.

Fortunately, problems of this kind do not occur frequently. It is gratifying that advances in thoracic surgery have made it possible to handle problems of this magnitude.

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RESTORATION OF THE THUMB

By Transplantation, Plastic Repair, and Prosthesis

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IN DESCRIBING losses of the thumb, certain terms are used, perhaps arbitrarily, and it might be well, therefore, to define these terms at the outset. A *partial loss* is one that involves no more than the distal phalanx. A *subtotal loss* involves approximately both phalanges of the thumb. A *total loss* means that not only are both phalanges missing, but that all or most of the metacarpal of the thumb is also absent.

PARTIAL LOSSES OF THE THUMB

Occasionally the amount of thumb loss is so slight that operative restoration is not practical. Much more frequent are cases in which there has been a loss of the distal phalanx which has healed with the formation of an adherent scar over the stump. In repairing such a condition, the surgeon is usually forced to choose between length of finger and preservation of highly specialized sensation. It is possible to lengthen the finger by utilizing a flap of skin, but if this flap is taken from a distant region (that is, not the skin of finger or palm), although in time it will develop primary sensation (heat, cold, pressure, pain), it will never develop stereognosis (see Figs. 1 to 3). Stereognosis, the highly refined perception which enables one, by touch alone, to distinguish and understand the form and nature of objects, might well be called the "eyes" of the fingers. It is dependent upon specialized touch corpuscles present in the digits and the palm. The pulps of the thumb and fingers, especially the first two fingers, are richly supplied with these corpuscles, but they are lacking in most skin areas of the body. Thus, if the tip of a thumb or finger is replaced by a skin graft or flap from a distant area, this fine perception can never develop.

A partial loss of the thumb which has healed with scarring on the distal and palmar aspects of the amputation stump therefore constitutes a troublesome problem. In such instances I believe it is ill-advised to attempt to replace the tip with a free graft or a distant flap. Occasionally flaps from the palm of the hand itself may be used successfully to replace a missing finger tip, but when a thumb is involved this is usually not feasible because of the difficulty of obtaining skin from the hypothenar eminence or base of the palm. It is my belief that, with rare exceptions, it is advisable in such a case to shorten the thumb and to use the normal palmar skin to replace the scar and cover the distal end of the thumb stump. This may even necessitate shortening the thumbnail.

Occasionally one encounters an individual who has sustained a partial or subtotal loss of the thumb, and whose index finger, although possessing normal sensation, is functionless because of bone, joint, and tendon damage. In such a case the end of the index finger may be transplanted to repair the thumb

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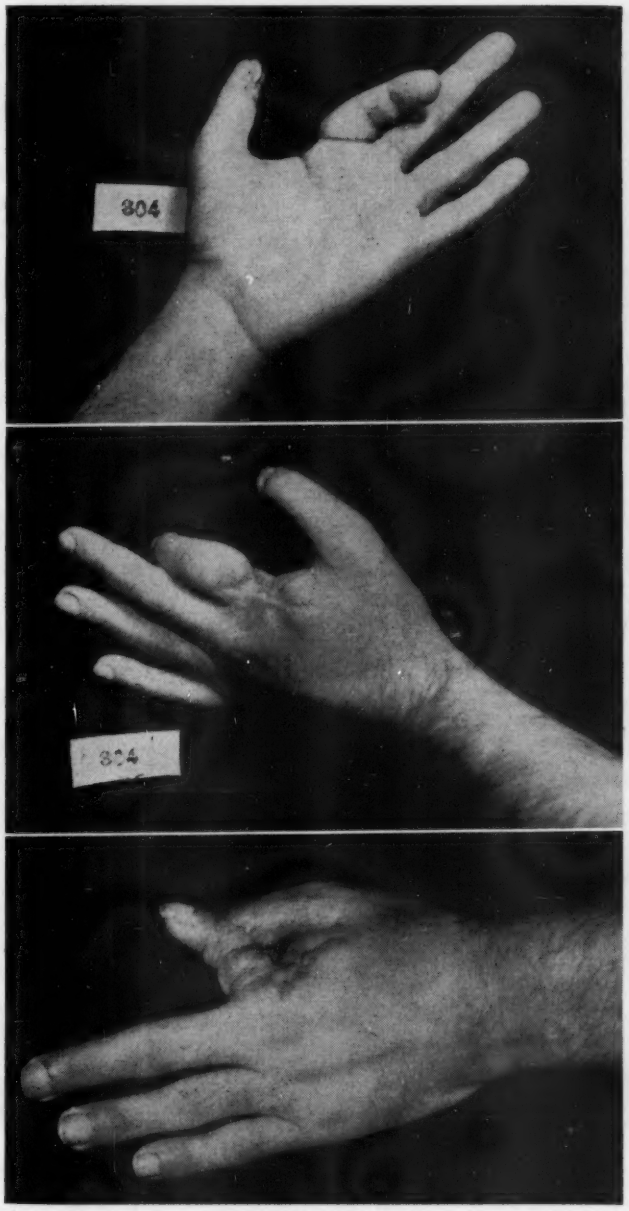
Fig. 1.—Partial loss of the thumb.

Fig. 2.—Abdominal flap migrated to the thumb in one stage. The pedicle of the flap is tubed and the donor area on the abdomen is closed. (Closed flaps of this nature, when properly planned and executed, are practically certain to take. They should be used in preference to open flaps.)

Fig. 3.—Final stage. The pedicle should be left attached to the finger for about three weeks. If there is any doubt as to the circulation, it may be tested by injecting sodium fluorescein intravenously and using ultraviolet light to observe the fluorescence. Clamping off the flap for a few minutes daily a few days before severing the flap is often useful.

(see Figs. 4 to 10). I had the opportunity of carrying out such a transplantation in the case of a young officer who had sustained a gunshot wound of the left hand.* The projectile had shattered the distal portion of the thumb and had



Figs. 4 and 5.—Palmar and dorsal views of a shrapnel wound of the hand. The distal phalanx of the thumb has been badly shattered and is covered with an unstable scar. There has been a compound fracture through the proximal finger joint of the index finger, with involvement of both flexor and extensor tendons. However, the index finger has normal sensation.

Fig. 6.—View illustrating the condition after healing of the first operation. The proximal two phalanges of the index finger have been fileted through a mid-dorsal incision and the index finger tip has migrated to the thumb after removing the useless tissue at the end of the thumb.

*This case was reported at the second annual meeting of the American Society for Surgery of the Hand, Chicago, Ill., January, 1947.

then passed through the proximal joint of the index finger, destroying the joint, the flexor and extensor tendons, and the skin covering. The circulation of the index finger was not affected and its digital nerves were also intact.

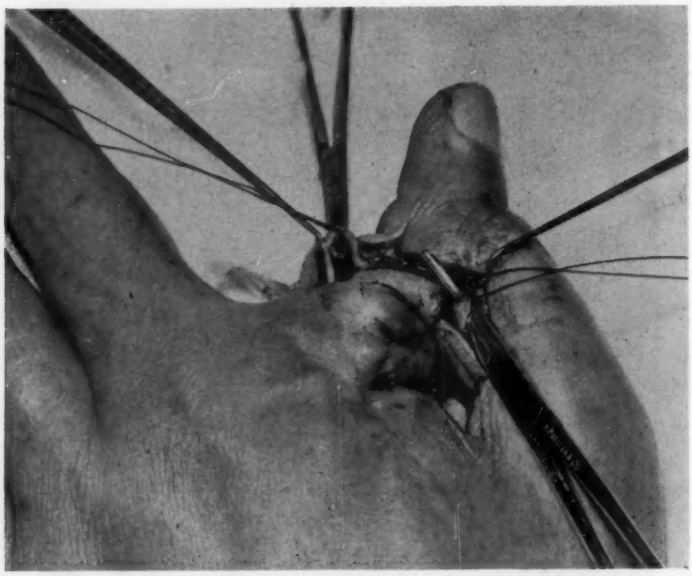


Fig. 7.—Photograph taken at the second operation, that of nerve suture. The digital nerve of the ulnar side of the thumb is elevated by the lower hemostat, while the upper hemostat shows the digital nerve on the ulnar side of the index finger. The nerves on both sides were united.

Fig. 8.—The thumb after the pedicle of the index finger was severed.

Since this index finger was practically useless, it was decided to transplant a portion of it to the thumb, to increase the usefulness of the thumb. All operative procedures were carried out under local anesthesia, procaine 2 per cent. At the first operation a middorsal incision was made on the index finger from the proximal to the distal finger joints. As this incision was made, the extensor tendon was reflected to one side, and the digital nerves and vessels were preserved. The bones of the proximal two phalanges were fileted, thus leaving a

tube of skin, its distal end consisting of the index finger tip with its phalangeal bone, digital vessels, and nerve. At this first operation the distal end of the thumb stump was freshened by doing a guillotine amputation through the distal joint. The distal end of the bone of the proximal phalanx itself was not disturbed. The long extensor tendon of the thumb was identified. The finger tip was then migrated to the thumb stump, and after uniting the extensor tendon of the index finger with that of the thumb, the remainder of the suturing was carried out as shown in Fig. 6.

I should like to emphasize at this point that the tendon suture was not carried out with the idea of restoring motion to this transplanted finger tip, but for the purpose of stabilizing the joint.

Fig. 9.—X-ray view taken before operation.

Fig. 10.—X-ray view taken after the transplantation had been completed. After this view was taken, the distal two-thirds of the second metacarpal was removed.

The migrated finger tip healed in place uneventfully. Approximately six weeks after the first operation, nerve suture was performed (see Fig. 7). Mid-lateral incisions were made on the pedicle of the index finger and the digital nerves exposed well proximal to the distal joint. In a similar manner, through mid-lateral incisions on the proximal phalanx of the thumb, the digital nerves of the thumb were exposed and dissected out distally to their ends. Fig. 7 shows the digital nerve of the thumb on one side and the digital nerve of the index finger on the other. Using 000000 black silk on atraumatic needles, nerve suture was carried out, uniting the proximal end of the digital nerve of the thumb to the distal end of the nerve of the index finger. The same procedure was, of course, carried out on both sets of digital nerves. The nerve suture was so located that it could be embedded in a healthy fat pad, and was located as

far distally as possible so that the new growth of axons would be as short as possible. At this same operation the digital arteries in the pedicle of the index finger were tied off in order to compel the finger tip to derive its entire circulation from the vessels of the thumb. It is no doubt possible to sever the pedicle at this stage, but I preferred not to do so in this instance. In approximately five weeks the patient developed a good return of sensation in the migrated tip. This sensation was, of course, associated as coming from the thumb tip.

The third operation was performed about six weeks after the second. At this stage the pedicle of the index finger was severed, and the flexor profundus tendon of the index finger sutured to the long flexor tendon of the thumb. Here again, it is emphasized that this was done for the purpose of stabilizing the joint, and not with the idea of giving motion to the finger tip. Fig. 8 shows the result obtained after the transplantation. Figs. 9 and 10 show x-ray views of the finger before and after the transplantation. The second metacarpal bone was trimmed down after this later x-ray picture was taken.

When last seen, in June, 1946, this patient had a stable, useful thumb, with good sensation.

I believe that a slight variation of this method could be used to obtain a movable joint. In order to do this, the joint of the index finger, with the distal part of the middle phalanx, would have to be transplanted after ablating the proximal half of the middle phalanx and the entire proximal phalanx of the index finger. Thumb amputation would then be carried out through the middle of the proximal phalanx. After migration and bony union had taken place, tendon transplantation could be carried out.

This method could also be used to transplant a finger from one hand to the other. Naturally one would hesitate to sacrifice a normal finger in order to reconstruct a thumb, but in the case here reported the index finger was useless.

Transplantation of the toe as substitute for a finger, following the method of Nicoladoni, is also a procedure which under certain special conditions might be attempted. Kuslik reported a successful case using Nicoladoni's procedure. The operation is best performed under local anesthesia. A transverse incision is made across the stump of the finger and its extensor tendon isolated. The distal end of the phalanx is exposed and the periosteum removed. An incision is made at the base of the second toe (the one generally used), and a skin flap formed after dividing both dorsal interosseous arteries. The extensor tendon of the toe is incised proximally as far as possible. A cuff of periosteum is turned back distally for about 1 cm. from the point at which the bone of the toe is divided. The distal end of the bone of the toe phalanx is dislocated into the wound and approximated to the freshened end of the bony stump of the finger. The periosteum of the toe is sutured to that of the finger, thus helping to maintain the bones in contact. The extensor tendons are then sutured in such a fashion that the suture line is proximal to that of the periosteum. The skin flap of the toe is then sutured to the skin of the finger and the wound closed on the palmar surface of the finger and the dorsal surface of the toe. A plaster bandage is then applied.

In Kuslik's case, immobilization was maintained for about one month. However, ten days postoperatively, pressure was applied to compress the plantar circulation and force dependence on the finger circulation. One month after the first stage, the second stage was carried out. The remainder of the toe was divided, the flexor tendons united, and the skin incisions closed.

Six technical details recommended by Kuslik are:

1. A plantar toe pedicle is advised because the plantar arteries are better developed than the dorsal. The patient will also be more comfortable if the finger is placed over the toe rather than under it. Both plantar arteries should be preserved.

2. The levels of the periosteal, tendon, and skin sutures should not coincide.

3. The periosteal cuff formed on the phalanx of the toe will facilitate better contact and immobilization of the bones.

4. The plaster bandage should be applied from the shoulder blade downward, covering the upper and lower extremities on the side of the operation.

5. The pedicle must not be severed too early.

6. The recipient must be kept warm.

I have had the opportunity of transplanting the toe only once, and in this particular case the result was not successful, because of three factors. First, the apposition between the finger and the toe was not close enough to fulfill the requirement of primary tissue contact. Second, the early stages of immobilization caused the patient such discomfort and she was so restless that there was a slight disturbance of the contact. Third, because of this discomfort the pedicle of the toe was severed, perhaps too early, on the sixteenth day.

SUBTOTAL LOSSES OF THE THUMB

In subtotal losses of the thumb where there remains a mobile metacarpal covered with a good scar, and where the patient is able to grasp, surgery may not be necessary. However, as a general rule, the prehensile function of the hand is much improved if the web is deepened. This may be accomplished quite simply by the Z-plastic operation, with transposition of flaps.

If conditions permit, subtotal losses of the thumb may be repaired by migration of another finger, as described previously. It is essential that such a restored finger have sensation, for without it the finger cannot function efficiently. The skin will become atrophic and may even ulcerate. A restoration of this type should therefore always include nerve transplantation and suture.

Phalangization of the thumb metacarpal is often very helpful in aiding the prehensile function of the thumb. This may be brought about by using either a flap or a free graft. My own preference is for a flap, because a flap will permit the use of heavier skin, which will be more durable than a free graft. Figs. 11, 12, and 13 show a case of this type. While it is desirable to obtain as deep a web as possible, muscular attachments should not be sacrificed. If they have already been damaged, a tendon transplant to bring about opposition, as described by Bunnell, is advocated.

TOTAL LOSSES OF THE THUMB

Total loss of the thumb, including loss of the metacarpal, reduces the efficiency of the hand by approximately 40 per cent. Such a hand lacks opposition and therefore cannot perform those functions which require prehensile movements. On the other hand, it is sometimes astonishing how much dexterity a patient who has lost the entire thumb may develop. Some patients, thus, do not wish to have any surgery done, nor do they desire a prosthesis.

The simplest form of prosthesis is basically somewhat similar to the "sailor's palm." Although the sailor's palm was not designed for prehensile movements, it can easily be seen that if the palmar portion is elongated, the fingers

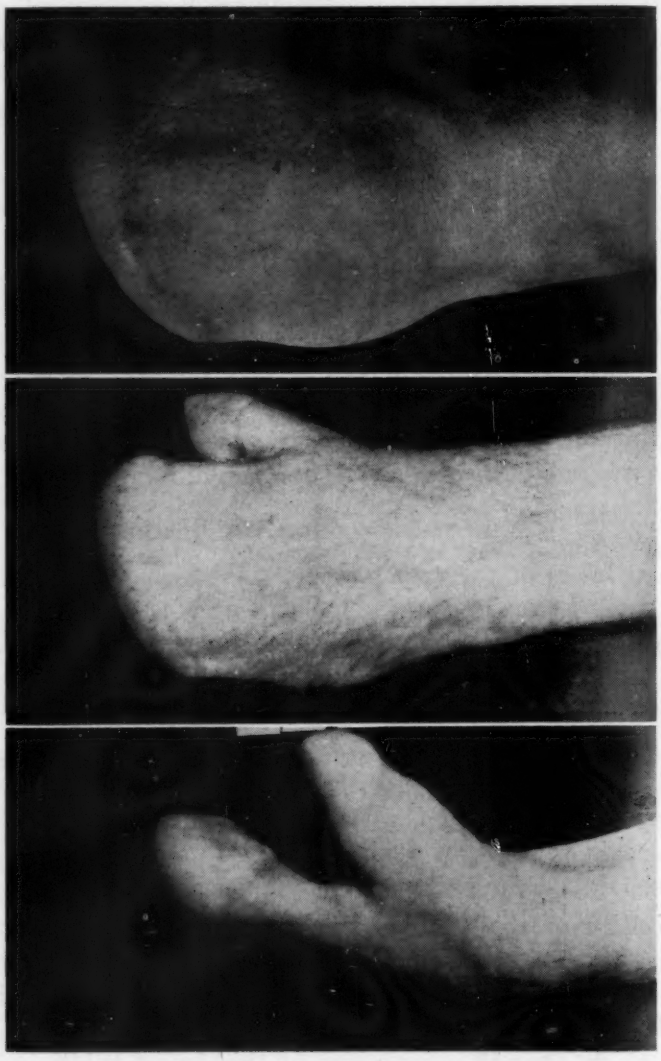


Fig. 11.—A loss of all five fingers. The metacarpals are all intact.

Fig. 12.—The result obtained by phalangization of the first metacarpal.

Fig. 13.—The amount of abduction present. This type of thumb reconstruction restores some of the prehensile functions of the hand, whereas previously the main member could be used only as a paw.

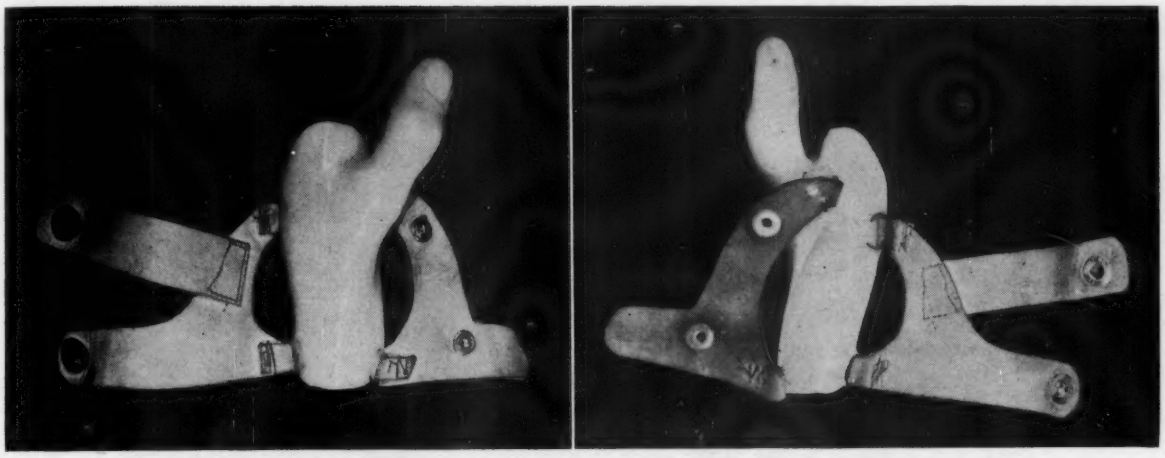


Fig. 14.—A working prosthesis. This thumb prosthesis is not to be confused with one which serves only to restore appearance.

Fig. 15.—Inside view of the prosthesis. Note that the palmar tip of the thumb is slightly roughened to prevent slipping.

Fig. 16.

Fig. 17.

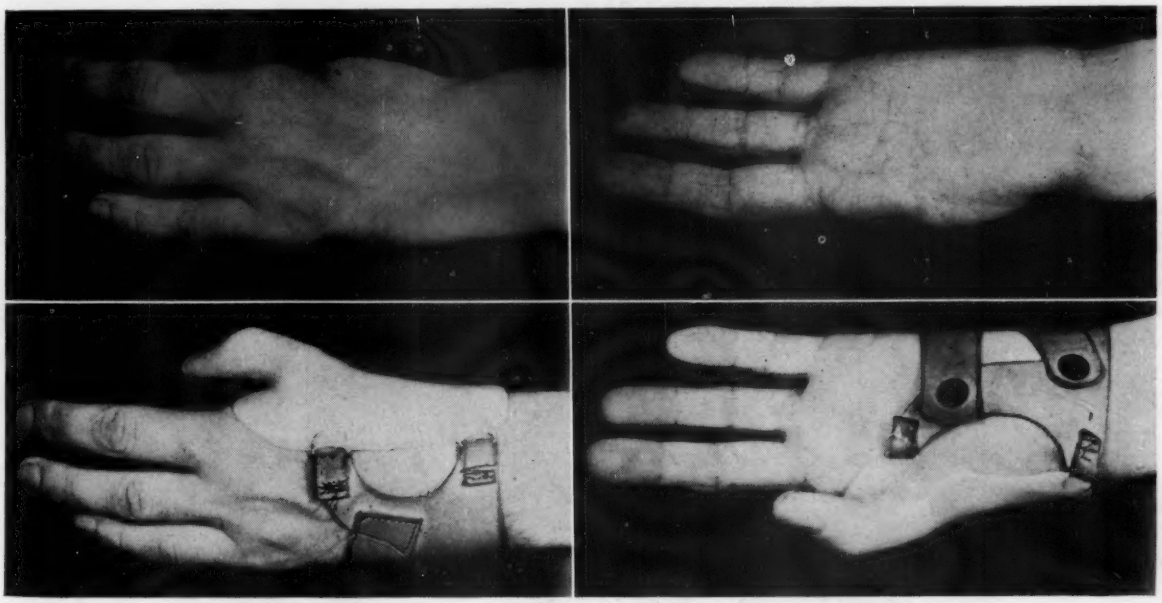


Fig. 18.

Fig. 19.

Fig. 16.—Loss of the thumb and index finger. Before a prosthesis can be used, the base upon which it is placed must be a stable skin covering with normal subcutaneous tissue. No sharp, bony spurs or irregularities should be permitted to remain, for they will only cause pain and discomfort, will interfere with the use of the prosthesis, and will discourage the patient.

Fig. 17.—A palmar view of the same hand.

Fig. 18.—The work prosthesis strapped in place.

Fig. 19.—A palmar view of the prosthesis in place. Note that snap fasteners have been used on the straps in preference to buckles.

may flex upon the post for grasping. The simplest type of prosthesis which we have used consists of a restoration made of acrylic resin, with its distal end slightly roughened to prevent slipping, and made with a sort of bracelet arrangement to attach the prosthesis to the hand. This thumb prosthesis is shown in Figs. 14 and 15. It is, of course, a work prosthesis, not a "dress" prosthesis. Its effectiveness is directly related to the patient's desire to use it. Those patients who have had the desire and the persistence to learn to use a prosthesis have found it very helpful (see Figs. 16 to 19). In this connection, it is well to

Fig. 20.—A curved lead pattern. This is sterilized and the graft fabricated according to it. The lead pattern is prepared beforehand and varies not only according to the size of the hand but also in length, depending upon the presence or absence of an index finger and upon the location of the recipient site.

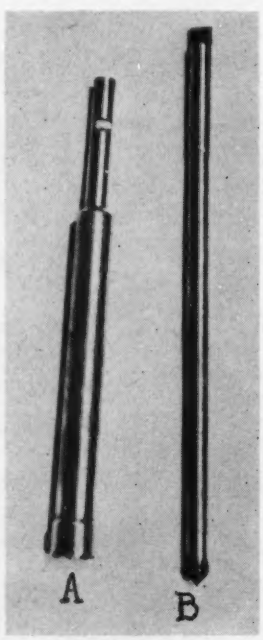


Fig. 21.—*A*, The peg-former; *B*, the drill used to prepare the donor site. The drill is marked off in centimeters. It cuts only at the end and therefore cannot become entangled with the soft tissue.

note that it is not wise to urge a patient to have a prosthesis made. On the contrary, it is best to explain carefully and honestly the advantages and disadvantages of the mechanical device, and at the same time to point out the possibilities and limitations of surgical restoration of the thumb. The patient who is permitted to make his own choice is much more satisfied with the result, and much more interested in developing skills, than the patient who reluctantly consents to corrective steps urged by the surgeon.

Surgical restoration for total loss of the thumb may be accomplished in a number of ways. The presence of even a small proximal portion of the metacarpal which has mobility greatly increases the usefulness of the surgical restoration. It may sometimes be possible, in restorations where the entire

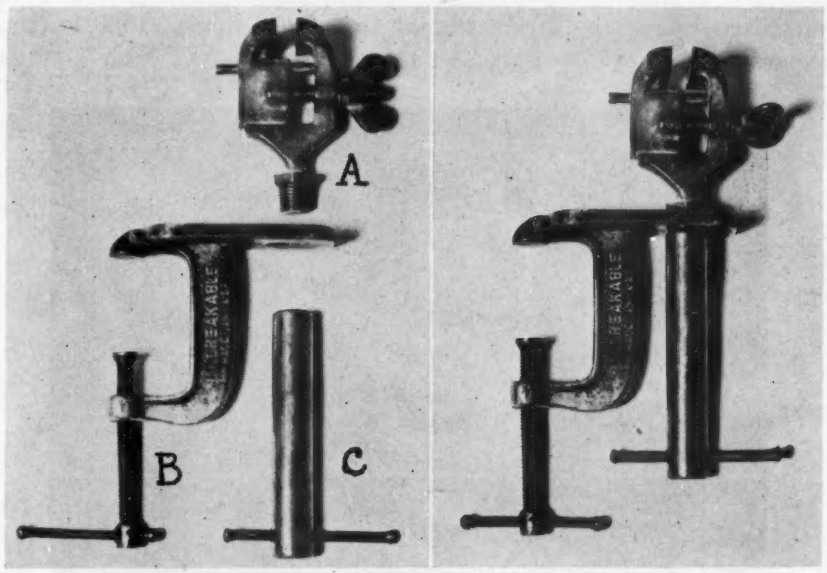


Fig. 22.—The component parts of the bone vise. When the vise head (*A*) is attached to the handle (*B*), the vise may be held in the hand.

Fig. 23.—The vise assembled so that it may be clamped to a table. By loosening the handle, the vise may be adjusted at any horizontal position.

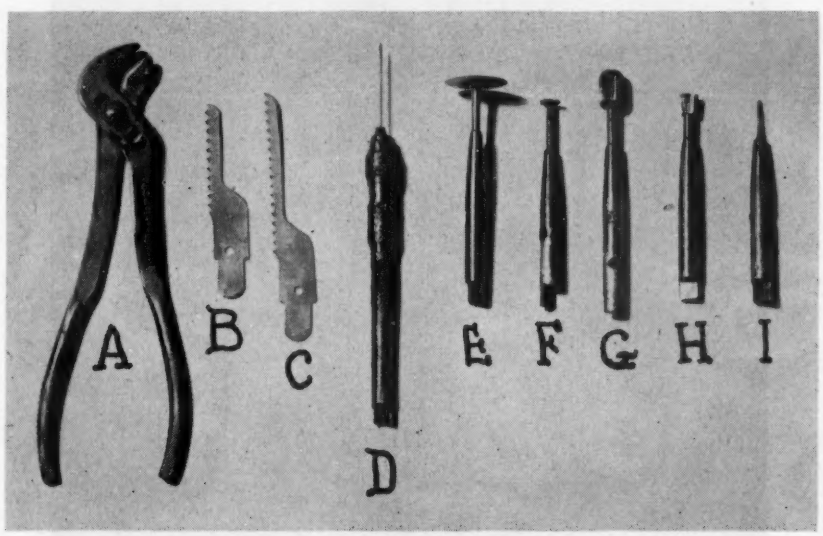


Fig. 24.—The shaping of the bone graft is expedited by the use of power instruments. *A*, A small bone-grasping forceps. These are mechanics' small ignition pliers. *B* and *C*, Small blades used in the reciprocating saw hand piece. *D*, The small chuck which fits into the rotary hand piece. This is used for driving in the Kirschner wires under power. *E* and *F*, Small circular saws used to cut and shape the graft. *G* and *H*, Rotary files. *I*, A fissure burr used to make the end cuts when taking a tibial graft. Note that these instruments fit directly into the rotary saw hand piece and avoid the used of a heavy, cumbersome chuck.

metacarpal is gone, to provide motion by making a false joint and carrying out tendon transfer.

If the index finger is present and if, after due consideration, it is thought advisable to use this finger to replace a lost thumb, the first step is to phalangize the metacarpal of the index finger. The index metacarpal is separated from the third metacarpal, but the structures of the index finger, that is, the flexor and extensor tendons, the vessels, and the nerves, are kept intact. It is neces-

Fig. 25.—Loss of the thumb and index and middle fingers following a hand-grenade explosion.

Fig. 26.—Palmar view. Note the thin, tight scar over the rough bony prominences.

Fig. 27.—Double exposure, showing the range of motion.

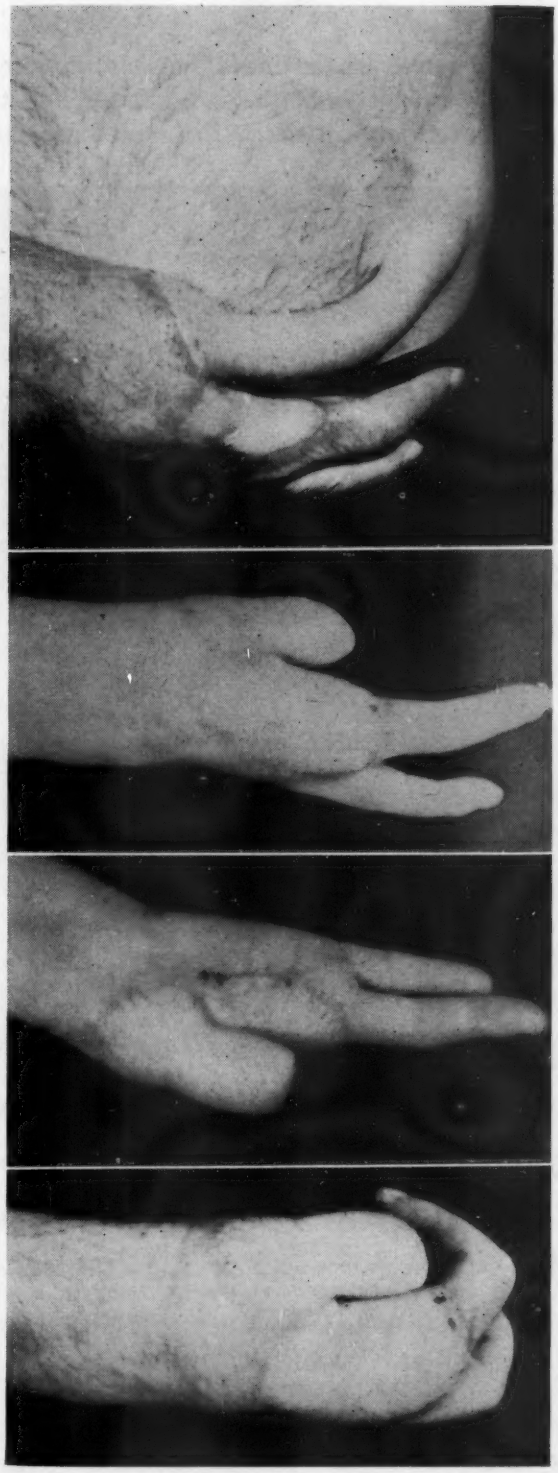


Fig. 28.—A tube pedicle has been migrated to the hand. This will be large enough to replace all the unstable skin. Before migration of the tube pedicle, rough bony spurs were removed.

Fig. 29.—Dorsal view after insertion of the bone graft.

Fig. 30.—Palmar view. Note that the flap has replaced all the unstable scar with heavy skin and good subcutaneous padding.

Fig. 31.—The range of motion after healing. It was necessary to release the flexor tendons of the ring finger to bring about this degree of flexion.

sary to split the digital nerve of the index finger from the common digital nerve which supplies the ulnar side of the index finger and the radial side of the middle finger. The raw area between the second and third metacarpals

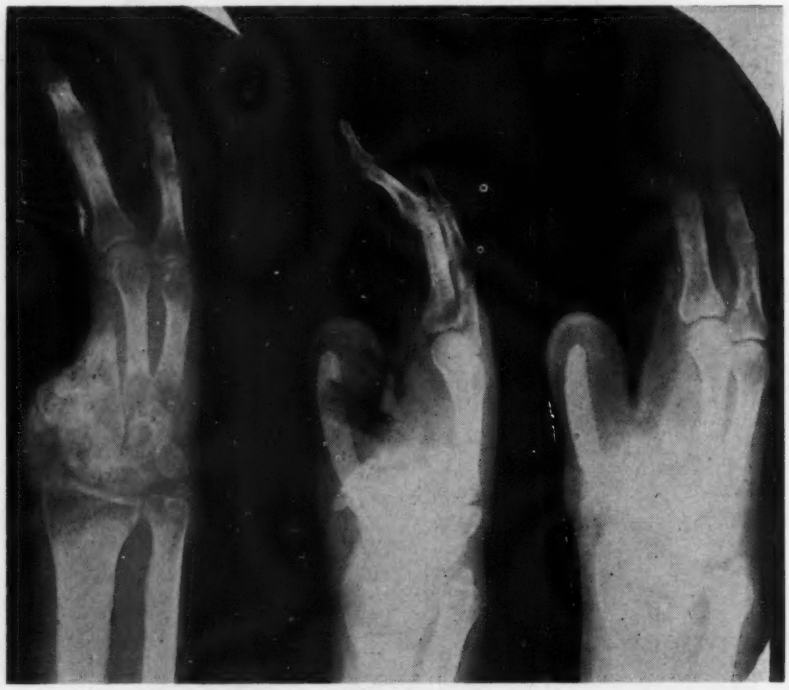


Fig. 32.—X-ray view taken before operation.

Fig. 33.—X-ray view taken after insertion of the bone graft. Curved iliac bone was used and fastened in place with two Kirschner wires.

Fig. 34.—Another postoperative x-ray view.

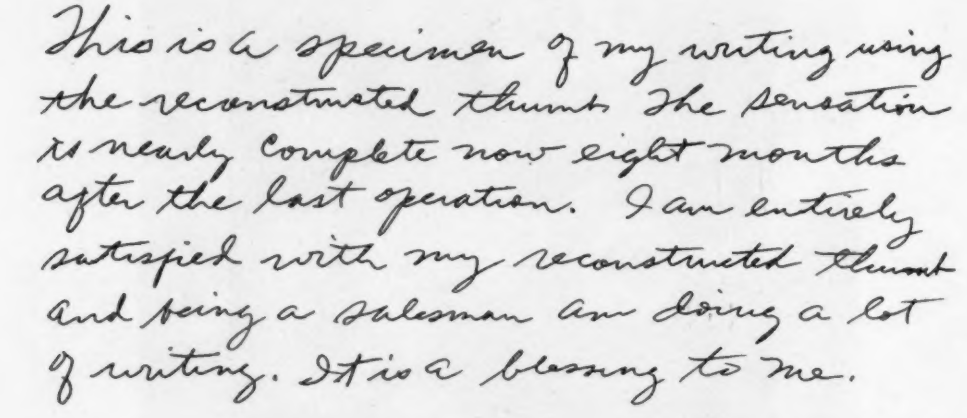

This is a specimen of my writing using
the reconstructed thumb. The sensation
is nearly complete now eight months
after the last operation. I am entirely
satisfied with my reconstructed thumb
and being a salesman am doing a lot
of writing. It is a blessing to me.

Fig. 35.—Specimen of the patient's handwriting made eight months after the final operation.

should be covered with a flap of skin. This flap should be of generous size to permit the migration of the metacarpal of the index finger to the position of the thumb. At a subsequent operation the index metacarpal is migrated to the

position of the thumb and so rotated that the index finger is placed in the position of opposition. At the same time the metacarpal should be shortened.

In those cases where it was considered desirable to provide a thumb, that is, simply an immovable post in the position of opposition, we have used a procedure which is well standardized. An abdominal tube pedicle is prepared at the first operation.* At a second operation, one end of the tube is migrated

Fig. 36.—Total loss of the thumb.
Fig. 37.—Tube pedicle attached to the thenar eminence.

Fig. 38.—Postoperative view from the radial side after the implantation of a straight tibial bone graft. The cleft is in line with the long axis of the forearm.
Fig. 39.—A palmar view. An attempt is made to locate the thumb in a position of opposition, the tip being opposite the cleft between the index and middle fingers.

to the thenar eminence. It is desirable to locate the scar in a neutral position, that is, away from areas of friction and use. At a third stage, the abdominal end of the pedicle is divided. Since the distal end of the tube is a terminal structure, it may be well to clamp off the pedicle prior to severing it. A rubber-covered clamp should be applied distal to the site at which the pedicle is to be divided. At the first trial, the clamp is applied only for a few minutes. If the portion of the pedicle attached to the hand does not become cyanotic, the pedicle may be clamped on succeeding days for longer periods of time. If there remains any doubt about the vigor of the circulation, it is best to sever the pedicle partially and to wait about one week before detaching it completely.

*It is possible to migrate the flap in one stage, tubing the pedicle, but I believe that if this is done in two stages, the flap has a much better circulation at its distal end.

A bone graft may be inserted in the pedicle at the third stage operation, but I have found it more desirable to implant the bone at a later stage. The reason for this is that the distal end of the tube may heal slowly, and if a bone graft has been inserted, there is a possibility that the skin will break down and

Fig. 40.—The same patient wearing the combined splint and protector. This simple device is made of a transparent synthetic resin.

Fig. 41.—Preoperative x-ray view.

Fig. 42.—Postoperative x-ray view showing the straight tibial bone graft immobilized by two Kirschner wires. If the wires cause no trouble and do not loosen, they may be left in situ, otherwise they are easily removed.

endanger the result. I have tried various sources for bone grafts: tibial bone, both straight and angulated; the curved portion of the ilium; and finally, a rib graft. I believe that the angulated or curved graft is most desirable, and that the iliac graft is preferable to the tibial, since the former heals more readily. The rib graft has no particular advantage.

The angulation or curve of the graft should be designed prior to the operation. Fig. 20 shows a simple lead pattern that is used to shape the graft. Fig. 21 shows a peg-former (*A*) and end-cutting drill (*B*).

Straight grafts from the tibia are cut with the twin-bladed rotary saw from the upper part of the anteromedial surface. The anatomic margins are avoided. A heavy rotary burr is used to cut the ends of the graft. When an angulated graft is taken from the tibia, the reciprocating saw is used. A curved graft taken from the crest of the ilium is removed by means of a wide, sharp chisel. When rib is used (preferably the eleventh or twelfth), it is removed in the usual manner, using Doyen rib elevators and rib shears.

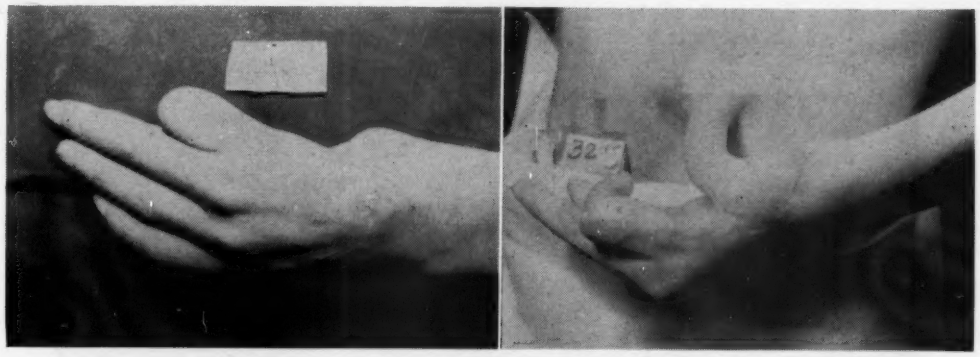


Fig. 43.—Another case showing total loss of the thumb. The bone graft in this case was on angulated tibial graft.

Fig. 44.—Tube pedicle migrated to the hand.

Fig. 45.—Postoperative result.

The placement of the bone graft requires great care, for unless the thumb post is in the proper position, its efficiency is markedly decreased. It should be so placed that the cleft between the post and the index finger, when viewed from the radial side of the hand, is aligned with the long axis of the forearm, and when viewed from the palmar aspect of the hand, the post should be in a position

of opposition to the cleft between the index and middle fingers (or to the radial and adjacent fingers if the index is missing). The post must not be too long, for in this case it will tend to interfere with the closing of the fingers. When the post is of the proper length, the patient may pinch against it with the index and middle fingers by *fixing* the two distal joints of each finger and moving the metacarpal phalangeal joints. When the post is to be cleared by the fingers, the two distal joints are flexed first, following which the metacarpal phalangeal joints are flexed.

When the proper site for insertion of the bone graft has been located, the drill is inserted into the carpal bones or into a remnant of the metacarpal if it is present. The drill is driven down to a predetermined depth; this is easily ascertained from the markings on the drill, as shown in Fig. 21, *B*.

Fig. 46.—Preoperative x-ray view.
Fig. 47.—Postoperative x-ray view showing the angulated tibial graft.

The shaping of the bone graft is expedited by the use of a simple bone vise and power-driven instruments (see Figs. 23 and 24). The general shaping of the bone graft is done with a rotary file. All sharp edges are smoothed and the distal end is carefully rounded. The graft is then placed in the bone vise, proximal end upward, and the peg-former (see Fig. 21, *A*) in the rotary saw handle is driven down on the graft to form a peg of the same diameter and length as the drill hole. The graft is then placed in the hole which has been prepared for it, and if it fits satisfactorily, it may be fastened in situ. All our grafts have been fastened with stainless steel Kirschner wire, using a simple,

Fig. 48.—Dorsal view of a hand in which the restoration was carried out by using a rib graft.

Fig. 49.—Palmar view of the same hand.

Fig. 50.—The reconstructed thumb. The thumb should never be made too long, lest it interfere with function.

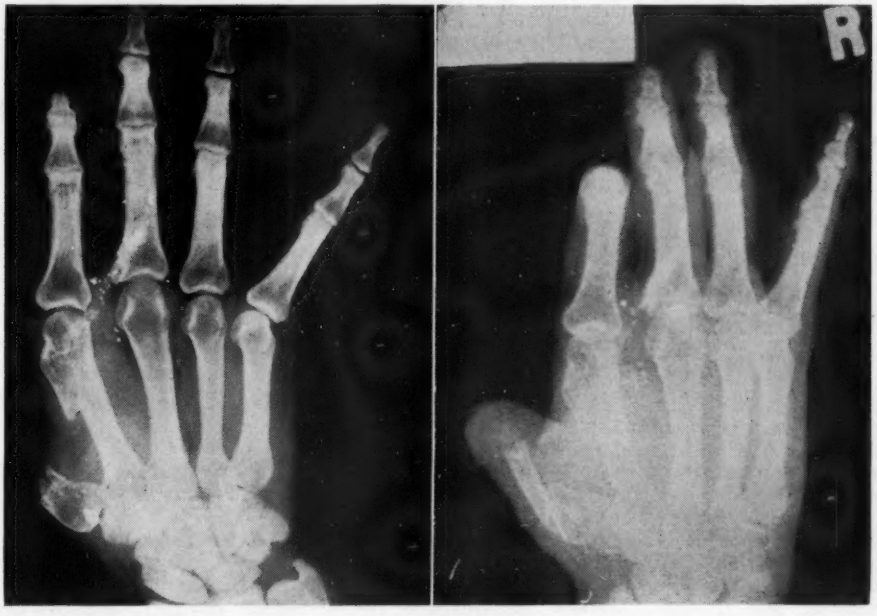


Fig. 51.—Preoperative x-ray view.

Fig. 52.—Postoperative x-ray view showing the rib graft.

efficient device to drive the wire in under power (see Fig. 24, *D*). Anyone who has attempted to use the conventional egg-beater type of hand drill to insert Kirschner wire in a small bone realizes how inefficient and laborious such a procedure can be. With a power-driven device, the placement of the wires can be carried out with ease and precision. Two wires are usually sufficient to hold the graft securely. The wires are left in indefinitely. However, if they should loosen, or if x-ray examination should show absorption around them, they should be removed.

The skin covering should be sutured carefully over the bone graft and a simple plaster splint applied to immobilize the thumb and wrist. This cast is removed after about three weeks and replaced by a protector, as illustrated in Fig. 40. This protector serves to immobilize the graft and to prevent injury to the skin of the restoration. It must be stressed that the development of sensation in the skin of the restoration may take as long as a year. During this time, the restoration may be damaged. Protection during the interval while sensation is developing is therefore important, and the patient should be cautioned in the care of the restored thumb.

Figs. 25 to 52 show a number of thumb restorations, using different types of bone grafts.

Complications.—In nine cases in which the thumb was restored, only one complication developed: ulceration of the distal end of the thumb before the implantation of the bone graft. This was attributed to early severance of the tube, without progressive clamping off of the circulation.

SUMMARY AND CONCLUSIONS

A successful new technique for restoring the thumb by means of a finger transplantation and nerve suture is presented. This technique is applicable to certain special cases which are discussed.

Other methods of restoration of the thumb in cases of partial, subtotal, and total loss are described and illustrated. The use of different types of bone grafts is discussed. A practical work prosthesis is shown.

Loss of the thumb is so crippling a disability that every effort toward restoration should be made. In properly selected cases, a carefully thought-out and well-executed restoration will result in a marked improvement in the functioning of the hand.

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TYPHOID OSTEOMYELITIS OF THE RIBS TREATED WITH STREPTOMYCIN AND SURGICAL EXCISION

A Case Report

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STREPTOMYCIN has been shown to be of great value in the treatment of certain infections, while in others its effectiveness is still in doubt.[1, 2] The sensitivity of many gram-negative bacilli to this new antibiotic has led to the hope that it will be useful in controlling typhoid infections. In vitro, *Eberthella typhi* is sensitive to concentrations of streptomycin readily obtained in the blood stream of patients receiving divided doses of 2,000,000 to 3,000,000 units daily.[3, 4] In some studies there has appeared to be definite improvement in patients with acute typhoid fever while in others the evidence of benefit is not clear cut.[1, 5] Streptomycin has had only a transient effect in reducing the number of organisms in typhoid carriers,[6, 7] and its use in other typhoid sequelae has not been reported.

Typhoid osteomyelitis is now rarely seen although Murphy,[7] in 1919, found that it occurred in nearly 0.82 per cent of 18,840 cases of typhoid fever. It has long been recognized[8] that typhoid bacilli may remain in the bones for many years before clinical evidence of osteomyelitis is present. In the typhoid era surgeons found that only radical operations were effective in controlling this lesion.

CASE REPORT

W. K., a white man, 73 years of age, was admitted to the Hospital of the University of Pennsylvania on Oct. 24, 1946. Chief complaint was a draining sinus in the right anterior part of the chest wall. The onset of the illness was in September, 1944, when the patient became ill with what was proved to be typhoid fever. His wife, who was discovered to be a carrier, was presumably the source of infection. The acute illness, with high fever, anorexia, and weight loss, lasted for two months, and he recovered sufficiently so that he considered himself to be in normal health. In late November, 1944, he noticed that there was a tender swelling over the anterior surface of the lower right part of the chest which gradually increased in size and became fluctuant. His physician aspirated this and obtained pus, which on culture was positive for *E. typhi*. A roentgenogram of the affected area showed no evidence of bone or lung involvement.

Since this lesion did not regress under conservative measures, the patient was then operated upon in another hospital. An incision was made over the right lower part of the chest and pus was drained. The distal ends of ribs eight, nine, and ten were exposed, and several necrotic fragments of bone were removed. The wound was packed and partially closed.

Subsequent to this operation the wound did not heal, and he was admitted to the same hospital one year later, in April, 1946. Through an incision in the same area more bone

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was resected, and a portion of the adjacent sternum, which had apparently been infected, was curetted. During this operation the chest cavity was accidentally entered. The wound was packed open as before. The patient was very ill following this operation, but he was soon convalescent. However, the wound did not heal, and it was still draining on admission to this hospital. There had been no cough, hemoptysis, or other evidence of pulmonary disease.

Physical examination disclosed a well-nourished man of about 73 years whose only significant findings were confined to the right lower anterior part of the chest. At the level of the anterior portion of the seventh rib there was a transversely placed sinus 6 cm. in length discharging yellowish-white pus. Immediately superior and medial was another small sinus. There were signs of underlying thickened pleura. A probe could be passed through the larger sinus medially and posteriorly for 4 cm.

Laboratory examinations were as follows: Hemoglobin, 72 per cent; white blood cells, 10,000. Urinalysis was negative. Culture of the wound yielded hemolytic *Staphylococcus aureus* and *E. typhi.* The nature of the *E. typhi* strain was further confirmed by the agglutination reaction. The Widal test was positive for *typhoid H* in a dilution of 1:640. Roentgen examination revealed that there were bands of adhesions and thickened pleura in the right costophrenic sinus. Lipiodol injection of the sinus showed no communication with the lung. Stool and urine cultures were negative for *E. typhi,* and on this organism streptomycin sensitivity tests showed that inhibition took place at concentrations of from 0.5 to 5.0 units per cubic centimeter. A culture taken from tissue at the time of operation after five days of streptomycin therapy was positive for *E. typhi.*

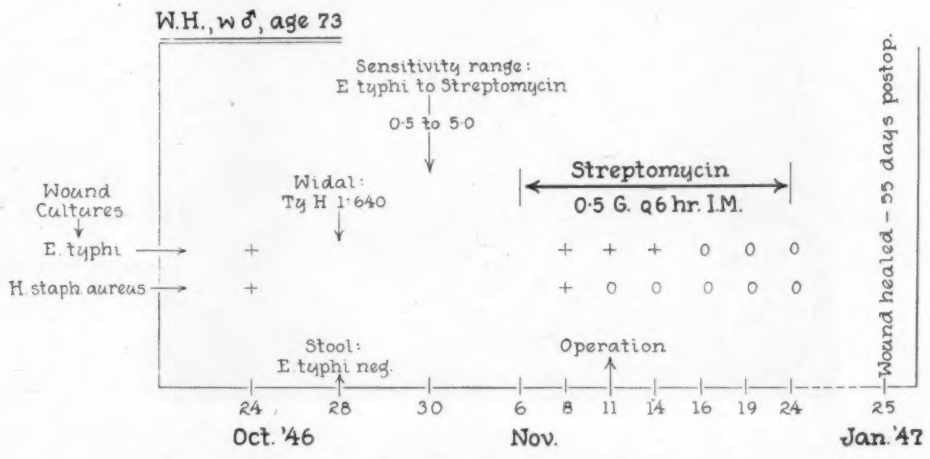

Fig. 1.—Protocol of patient with typhoid osteomyelitis of the chest wall.

Course.—Streptomycin administration was begun on Nov. 6, 1946, five days before operation and continued until Nov. 23, 1946, thirteen days after operation. One-half gram of the drug was given every six hours intramuscularly, and the total amount was 36.0 Gm., or 36,000,000 units, injected over a period of eighteen days. On Nov. 11, 1946, eighteen days after admission, extensive resection of the diseased area in the right lower chest wall was performed. The draining sinuses and associated scar were completely excised through a long transverse incision and a shorter vertical one in the form of a "T" placed parallel with and adjacent to the sternum. The costal cartilages and anterior bony ends of ribs five to ten, inclusive, were excised. The right lateral margin of the sternum was also resected, along with the upper portion of the rectus sheath. An en bloc dissection was carried out, and normal tissue surfaces remained in the wound everywhere except at the center where thickened pleura attached to the lung was only partially removed. The entire cavity was packed with iodoform gauze and the skin edges approximated except for two openings where the ends of the packing protruded. Considerable bleeding took place, estimated at over 500 c.c., and this was replaced by blood transfusion during the procedure.

The postoperative course was satisfactory. The wound drained a large amount of serosanguineous material, and the packing was completely changed on the fifth day after operation. On Nov. 20, 1946, nine days postoperatively, enough granulations had appeared to make packing unnecessary. At this time the wound was free of pus and necrotic tissue. Cultures of the wound showed that *E. typhi* had disappeared by the fifth postoperative day and had not returned up to the day of discharge, November 24, thirteen days after operation.

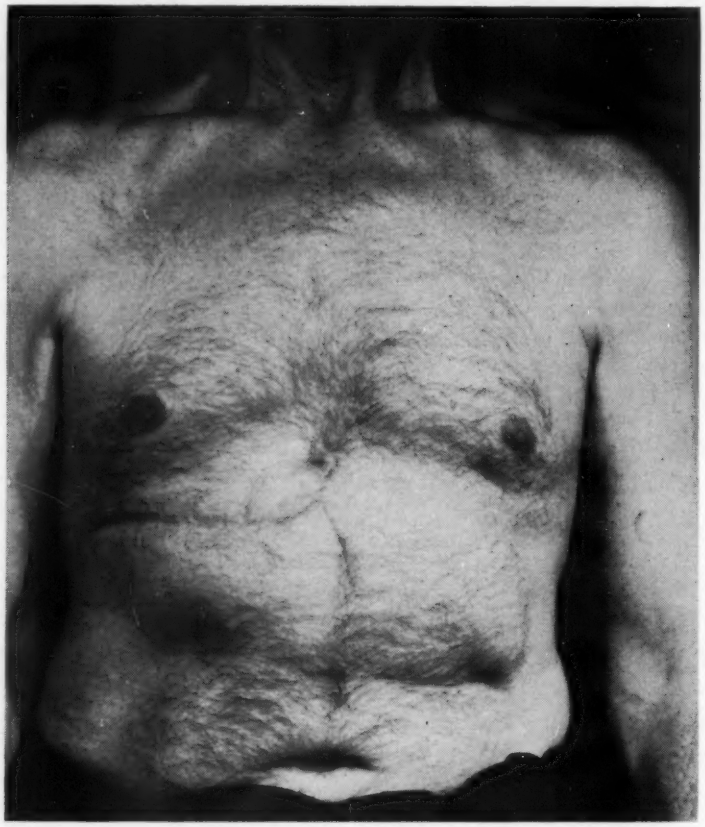

Fig. 2.—Patient W. H., with typhoid osteomyelitis of right anterior part of chest wall. End result five months after radical operation and streptomycin therapy.

The patient was followed in the outpatient clinic at intervals for redressing, and it was noted that the wound had healed over completely by Jan. 5, 1947, fifty-five days after operation. By April, the wound had remained closed for three months, and the patient was entirely asymptomatic.

COMMENT

Keen,[9] in reviewing his extensive experience with typhoid fever and its sequelae, concluded that "the tendency to chronicity, to persistent sinuses and especially to recurrences, are among the most marked characteristics of bone disorders following typhoid."

The sensitivity to streptomycin of the typhoid bacillus isolated from this patient suggested that the likelihood of cure following operation might be enhanced by the use of streptomycin, as in the use of penicillin for staphylococcal osteomyelitis.[10] Because of the chronicity of this lesion and the presence

of sequestra and fibrosis, cure or marked improvement was not expected with streptomycin alone. During the five-day interval of streptomycin therapy before operation there was no change in the quantity or quality of the purulent wound drainage, and typhoid bacilli were cultured from the chest wall.

SUMMARY

1. Streptomycin may be a valuable adjunct to the surgical treatment of typhoid osteomyelitis.

2. The use of streptomycin in one patient with typhoid osteomyelitis suggests that it will not replace adequate excision, just as penicillin does not replace surgery in the treatment of staphylococcal osteomyelitis.

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EXPERIMENTAL OBSERVATIONS IN THE TREATMENT OF CRANIOSYNOSTOSIS

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INTRODUCTION

ANY abnormality, whether congenital or acquired, which interferes with unrestricted growth of the human brain during the first years of life, results in corresponding interference with the neurologic functions which are elaborated during the same period. A large group of clinical entities has been described, exhibiting varying degrees of cranial deformity, amblyopia, and mental deficiency, whose common underlying pathology is restriction of normal brain growth due to premature fusion of the cranial sutures. These bony lesions may be grouped together under the term "craniosynostosis." Although the etiology of this condition is poorly understood,[1-3] the actual pathologic physiology, as a primary mechanical disturbance of cranial growth with secondary compression or distortion of the central nervous system, is generally recognized.

In the normal human skull, the separate bones of the calvarium begin to fuse with one another in the third and fourth decades of life and are usually not completely fused until the seventh or eighth decades. Normally, only the suture between the two frontal bones (metopic) fuses before birth or during the first few years of life. Occasionally this suture persists for a longer period, but as such is of no clinical significance.

In craniosynostosis, one or more of the sutures becomes prematurely obliterated, due presumably to some inherent mesenchymal defect rather than to any known pre- or postnatal disease process. The coronal and sagittal sutures are the most frequently involved and are of greatest clinical significance.

This abnormal fusion may occur before birth or during the first months of life. During the first six months the brain normally increases about 85 per cent in weight, and during the first year about 135 per cent.[4, 5] In the first six months more than 40 per cent, and during the first year more than one-half, of all postnatal increase in the circumference of the skull normally occurs.[5] It is during this first year of life, therefore, that unrestricted growth is particularly important.

TREATMENT

Craniosynostosis, because of its mechanical nature and apparent lack of primary pathology in the nervous system, suggests itself as a favorable and challenging lesion for surgical therapy. A number of different surgical procedures have been attempted to relieve the constriction caused by premature synostosis. Linear craniectomy,[6, 7] subtemporal decompression on one or both

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sides,[8, 9, 10] elevation of large bone flaps on one or both sides,[11] removal of plaques of bone,[12] creation of artificial sutures,[13] and "morcellation" of the entire calvarium above the cerebellar region[14, 15, 16] have all been employed. These operations have met with a certain amount of success in the relief of intracranial pressure and probable preservation of function in older children. However, during the first months of life these procedures would appear to be unsatisfactory because of the extremely rapid regrowth of bone which fuses artificially created defects at this age.

If unrestricted growth of the brain for an adequate length of time is to be insured in infants with craniosynostosis, repeated decompression or repeated creation of artificial sutures must be carried out. It has seemed desirable, therefore, to devise a method of producing artificial channels similar in distribution to the normal sutures which have prematurely fused, in which fusion could safely be delayed or indefinitely prevented. Such a means would then sustain during continued growth the immediate decompression obtained at the time of operation.

Theoretically, at least, one should attempt to reproduce as nearly as possible the normal pattern of growth. The possibility, therefore, of creating artificial sagittal and coronal sutures and preventing their subsequent closure for prolonged periods and perhaps indefinitely has commanded our attention. Since new bone arises from the outer layers of the dura as well as from the periosteum and eventually fills in any defect in the calvarium of young infants, it would appear necessary to introduce some substance between the margins of an artificially created suture, which would either delay this regeneration or prevent solid union of the new and old bone.

Any form of living tissue introduced into the gap could lead to formation of scar tissue, with resultant solid fibrous union, in which new bone formation could follow the invasion of osteoblasts from the bone margins. It seemed more profitable to investigate the possibility of introducing an inert foreign substance which would mechanically prevent the closure of such an artificial suture. Such a foreign substance should be well tolerated by bone as well as by dura and scalp; it should be light, suitable for sterilization, easily shaped, and resistant to undesirable physical or chemical changes when buried in the body.

EXPERIMENTAL INVESTIGATIONS

The problem first approached in the laboratory was to study the reaction of inert foreign substances placed between the margins of artificially created channels in the cranium. The following substances were investigated:

1. Fibrin film (obtained from the fractionation of human blood plasma)
2. Oxidized cellulose gauze (Oxycel)
3. Tantalum
4. Methyl methacrylate (Lucite)
5. Polyethylene (Polythene)

Observations on eleven dogs and sixteen monkeys are included in this study. Most of these animals were used for other experimental procedures

as well, but in no instance did the other experiments involve the cranium or its contents. Employing aseptic technique, midline incisions under intravenous nembutal were made, and the scalp was reflected to expose the vertex. Burr holes were made in the frontal region and in the postparietal region, approximately 1.5 cm. to either side of the midline. The frontal and postparietal burr holes on each side were connected by an ordinary saw cut, made with a standard Gigli saw. The saw cuts averaged 2.5 to 3.0 cm. in length. The edges of the saw cut were trimmed to right angles, by removing any roughened edges of bone with a sharp scalpel. This resulted in a gap averaging 2 mm. in width. Bone wax was used sparingly to control bleeding. Drill holes were made with a 1 mm. dental drill about 3 mm. from the lateral edge of the saw cut for the

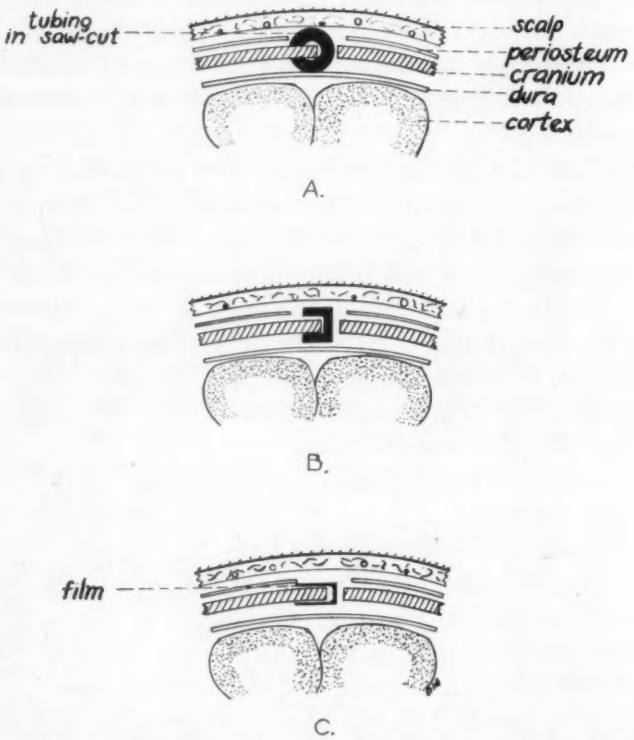

Fig. 1.—Schematic cross-section representation of the methods used to implant inert foreign substances into artificial sutures in the cranium of experimental animals. *A*, Tubing (polyethylene, fibrin film) inserted over bony margin of the saw cut; *B*, "U"-shaped insert (tantalum, Lucite); *C*, thin plastic film (polyethylene).

passage of 4-0 silk sutures. Two sutures were used in each case to hold whatever substance was introduced in the saw cut in position. On the right side of each animal, one of the substances just listed was introduced into the gap. On the left side, a saw cut of similar length and width was made in a similar location as a control (Figs. 1 and 2). The dura was not opened in any instance.

The monkeys were all small adults *(Macacus rhesus)* in good health with the exception of two animals who died early, presumably of tuberculosis. Among the monkeys there was no postoperative infection. Among the dogs there were two instances of superficial wound infection. Both adult and very young dogs

were used. Specimens were studied at intervals varying from 32 up to 315 days. Observations of the gross appearance of the foreign substance, the bone, overlying scalp, and underlying dura were made at the time of death or sacrifice in each case. Histologic studies were made of cross sections through the saw cuts containing each type of substance and through selected controls.

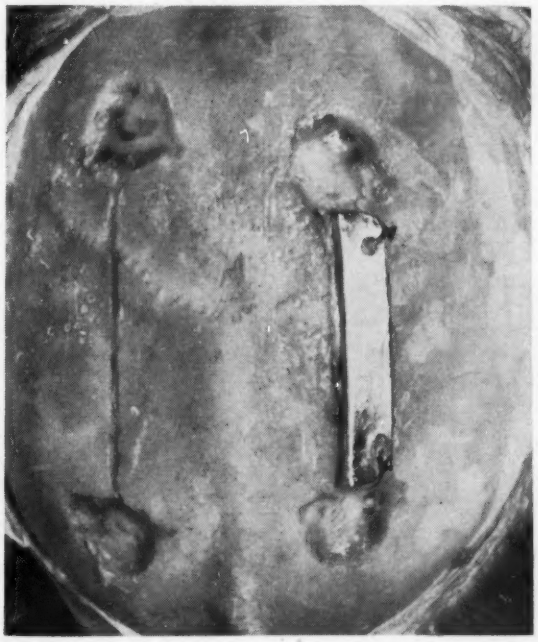

Fig. 2 (Monkey 57).—Appearance of the cranium thirty-nine days after insertion of a piece of tantalum over one margin of a saw cut. There is no evidence of new-bone growth or foreign body reaction. This figure illustrates the manner in which various inert substances were implanted in artificial cranial channels. Control saw cut is shown on the left.

RESULTS

Controls.—In the monkeys, the control saw cuts showed bony fusion in all those examined after 100 days. Of those examined before 100 days, one at thirty-nine days showed no bony fusion; one at seventy-five days showed complete closure, and another at ninety-one days showed only partial bony union. The remaining thirteen control specimens, ranging from 114 to 315 days, showed solid bony healing. Microscopic examination of the control saw cuts was made in several instances. Illustration of the healing in 114 days is shown in Fig. 3.

In the dogs, control specimens were examined at periods varying from 32 to 118 days. In every instance there was bony healing across the control saw cut. In young puppies the healing was particularly rapid. In most instances, in both the dogs and the monkeys, the burr holes also were partially or completely bridged by thin layers of new bone in specimens examined after 100 days.

Fibrin Film.—Fibrin film, made from pure fibrin derived from the fractionation of human blood plasma, has been shown by Ingraham, Bailey, and Cobb[17] to be well tolerated in experimental animals and in human patients.

They also demonstrated that this film was eventually completely absorbed. The possibility of delaying the closure of artificial sutures by interposing several thicknesses of fibrin film was therefore examined. Tubes were made by wrapping the fibrin film around glass rods of various sizes under sterile conditions. The wall of the tubing varied in thickness according to the number of layers of wrapping, usually 1 to 2 mm. At operation the tube was slit longitudinally and slipped over one margin of the saw cut, as shown in Fig. 1, *A*. It was held in position by two silk sutures, as previously noted. Fibrin film was implanted in this manner in three monkeys, and the specimens examined after 207, 209, and 257 days.

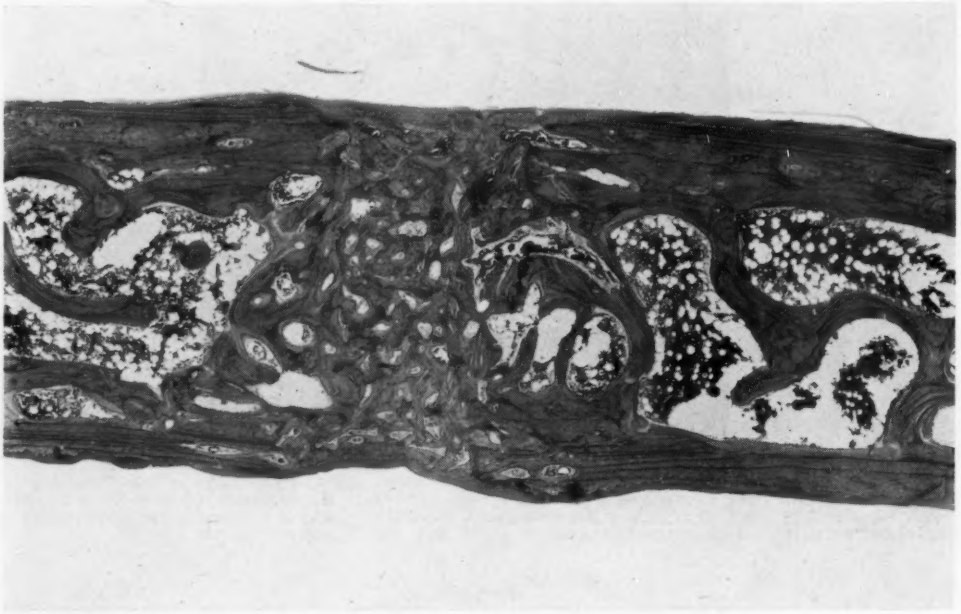

Fig. 3 (Monkey 53).—Low-power photomicrograph (×20) of cross section through control saw cut at 114 days. Solid fusion by dense new-bone formation across the gap has taken place.

In each instance there was no evidence of fluid and no gross evidence of foreign body reaction at the time of sacrifice. No trace of the fibrin film could be made out. In one of the specimens—that examined in 209 days—the saw cut was bridged by bone which was very thin compared to the new bone bridging the control saw cut. In the other two specimens, solid bony union of the saw cuts was present, no difference between this side and the control saw cut being discernible grossly.

Microscopic sections revealed no trace of the fibrin film. No evidence of foreign body giant cells or round-cell infiltration could be made out. The gap between the cut edges of the bone was filled with dense fibrous tissue in which osteoid tissue and new bone had formed (Fig. 4).

Conclusion.—Although several layers of fibrin film introduced into a saw cut possibly produced slight delay in bony union as compared with the control

saw cut, there was essentially no difference at the end of six to seven months. There was no unusual tissue reaction to the fibrin film.

Oxidized Cellulose Gauze.—Frantz and Lattes[18, 19] found that Oxycel—oxidized cellulose gauze—introduced experimentally into fracture sites in long bones prevented or markedly delayed callus formation; this occurred only when the gauze was soaked with blood when it was inserted, not when it was dry. Because of this finding, it was thought desirable to test this substance in artificial saw-cuts in the cranium. Consequently, blood-soaked Oxycel was introduced into the gap of saw cuts made in the cranium of four puppies. These specimens were examined in forty-two to forty-four days. At this time no gross evidence of residual Oxycel could be demonstrated. There was no gross inflammatory or unusual foreign body reaction demonstrable. There appeared to be solid fibrous union, which was grossly as strong as the early bony fusion on the control side.

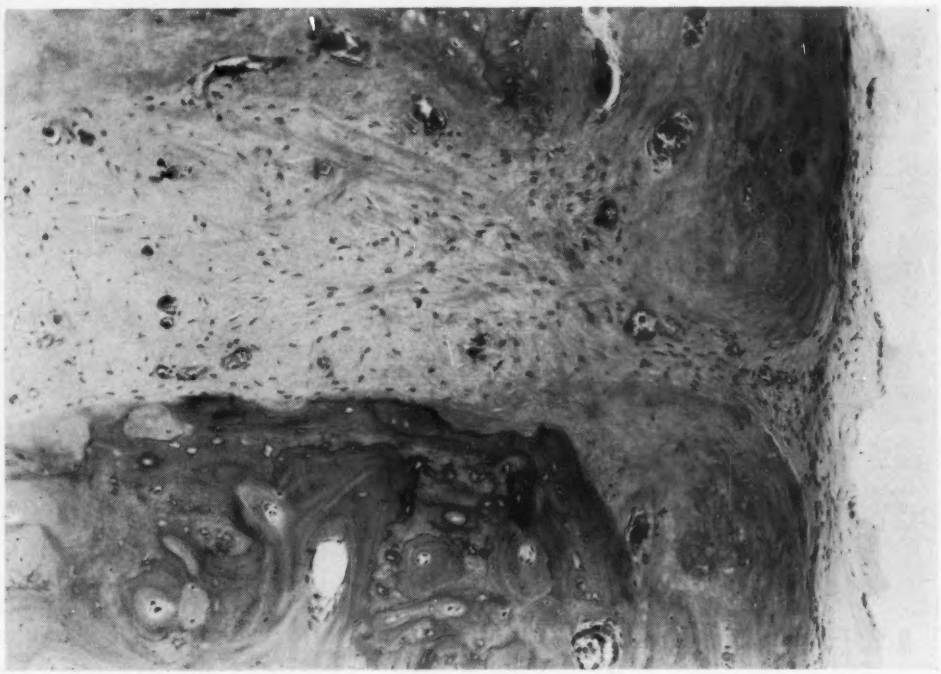

Fig. 4 (Monkey 58).—Photomicrograph of the periosteal margin of a cranial saw cut 207 days after insertion of several layers of fibrin film showing osteoid tissue invading the dense fibrous tissue in the gap. No trace of the fibrin film remains.

Microscopic sections showed the gap between the margins of the bone filled with dense fibrous tissue. Occasional small spaces which had been occupied by Oxycel were still present, and adjacent to them foreign body giant cells, young fibroblasts, and new capillaries could be found (Fig. 5).

Conclusion.—Because of the early dense fibrous healing across the saw cut, almost as rapid as that seen in the controls of the same length of time, it was felt that further experimentation with this substance as a material for delaying closure of artificial sutures was not indicated.

Tantalum.—The element tantalum has received an ever-increasing use in surgery during the past decade. During World War II, its application in the closure of cranial defects was developed into a standard, widely employed procedure. Its inertness in the body, and particularly its tolerance by bone, has been well established.[20, 21, 22] Tantalum can be formed in thin sheets which are malleable, but difficult to shape easily at the operating table. Tantalum is a heavy substance, opaque to x-ray, and expensive.

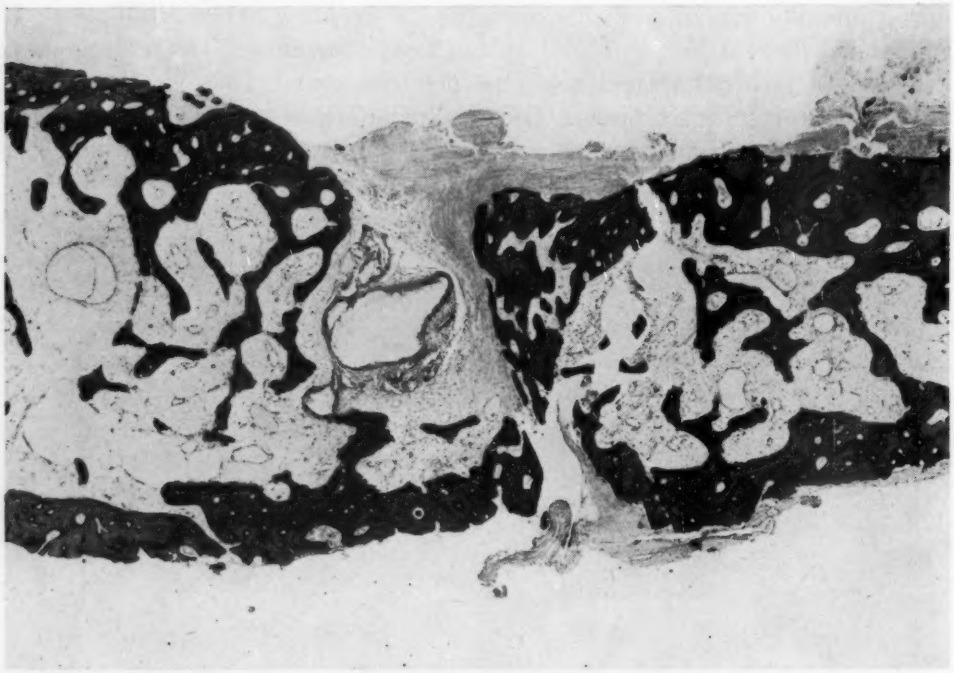

Fig. 5 (Dog 141).—Low-power photomicrograph (×20) of cross section through saw cut forty-two days after implantation of blood-soaked oxidized cellulose gauze. In the middle of the gap between the bony margins a space is visible in which the remains of the oxidized gauze were located. Dense fibrous tissue has invaded the gap. Grossly, this fibrous union was as solid as the control on the opposite side of the cranium.

In five monkeys, tantalum was inserted into the gap between the margins of a cranial saw cut. A piece of tantalum plate .015 in. in thickness was used, fashioned in the shape of a "U," the arms of which were approximately 4 mm. in width, the base a little more than the thickness of the cranium, that is, approximately 3 mm. This U-shaped piece of tantalum was inserted over the lateral margin of the saw cut, in the manner shown in Figs. 1, *B* and 2. It was held in place with two silk sutures. Specimens were examined after 39, 75, 91, 248, and 289 days.

Gross examination of these specimens in every case revealed no evidence of infection or foreign body reaction. There was a thin, filmy, fibrous tissue envelope around the tantalum, which did not contain any fluid. The inner surface of this envelope was smooth and glistening. It extended between the tantalum and the bone, between the tantalum and the dura, and between the

tantalum and the scalp. There was no evidence of new bone formation in this fibrous envelope.

Microscopic examination of cross sections through the saw cut after removal of the tantalum showed the thin, densely packed fibrous tissue membrane which completely surrounded the tantalum with a smooth, serosa-like inner surface adjacent to the metal (Fig. 6). Blood vessels were found in the deeper layers of the membrane. At the angles of the saw cut new bone formation had taken place, with many osteoblasts in evidence, but no evidence of any bone growing across the gap around the tantalum, either on the periosteal or dural surface (Fig. 7). It was noted that the membrane on the outer surface of the tantalum was thinner than that between the tantalum and the bone.

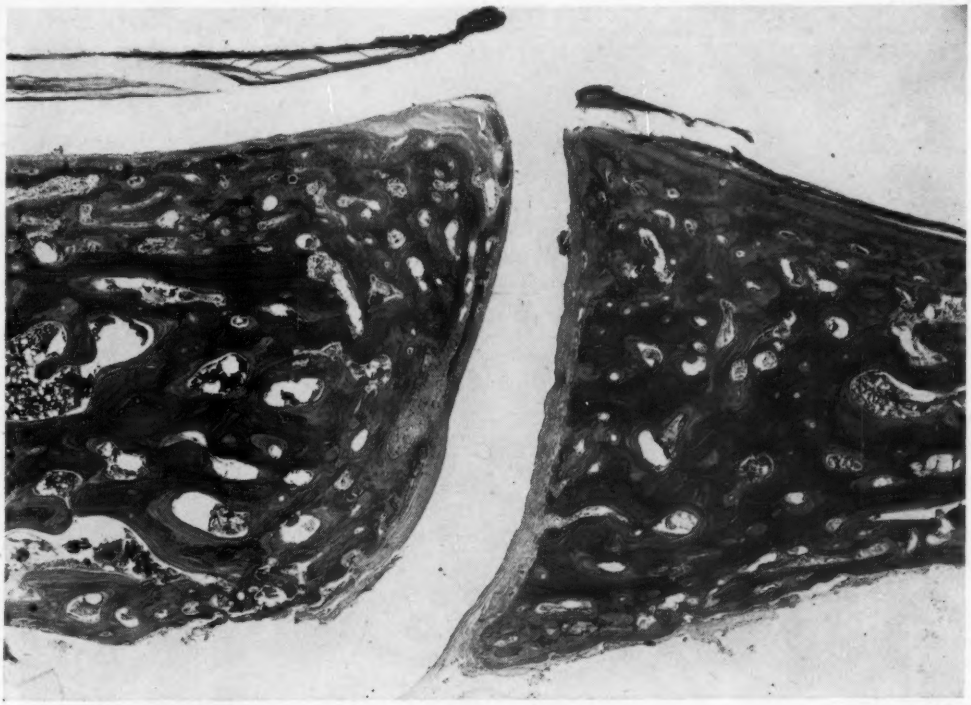

Fig. 6 (Monkey 59).—Low-power photomicrograph (×20) of cross section through saw cut in which a U-shaped piece of tantalum had been inserted over one margin 248 days previously. The fibrous tissue membrane between the bone ends and the site of the tantalum is easily visible. New bone formation at the margins of the saw cut has taken place but there is no invasion of the membrane over the tantalum.

Conclusion.—The tolerance of tantalum by bone and other tissues in the body has been confirmed. A nonadherent fibrous tissue envelope surrounded the metal. There was no attempt at new bone formation across the foreign body to bridge the saw cut. This substance would, therefore, seem suitable for permanent implantation. It is possible that the use of annealed tantalum foil might obviate some of the undesirable technical properties of tantalum plate. However, its weight and opacity to x-rays probably make tantalum a less desirable substance than others investigated.

Methyl Methacrylate (Acrylic Resin, Lucite, Plexiglas).—Methyl methacrylate, a synthetic plastic, has been shown, like tantalum, to be well tolerated in the tissues of human patients and experimental animals.[22-29] It has been used largely in the making of dental prostheses, and more recently in cranioplasty and other conditions where a rigid substance was required for burial within the body. Methyl methacrylate is a transparent, hard substance, which can be cast into desired forms. It can subsequently be molded to a slight degree by heating to temperatures just below that of boiling water. However, it is difficult to manipulate to any considerable degree once it has been cast in a given shape. It is light, nonopaque to x-rays, and relatively inexpensive. Its lack of undesirable reactions in contact with bone has been demonstrated repeatedly, as pointed out by Woolf and Walker.[22]

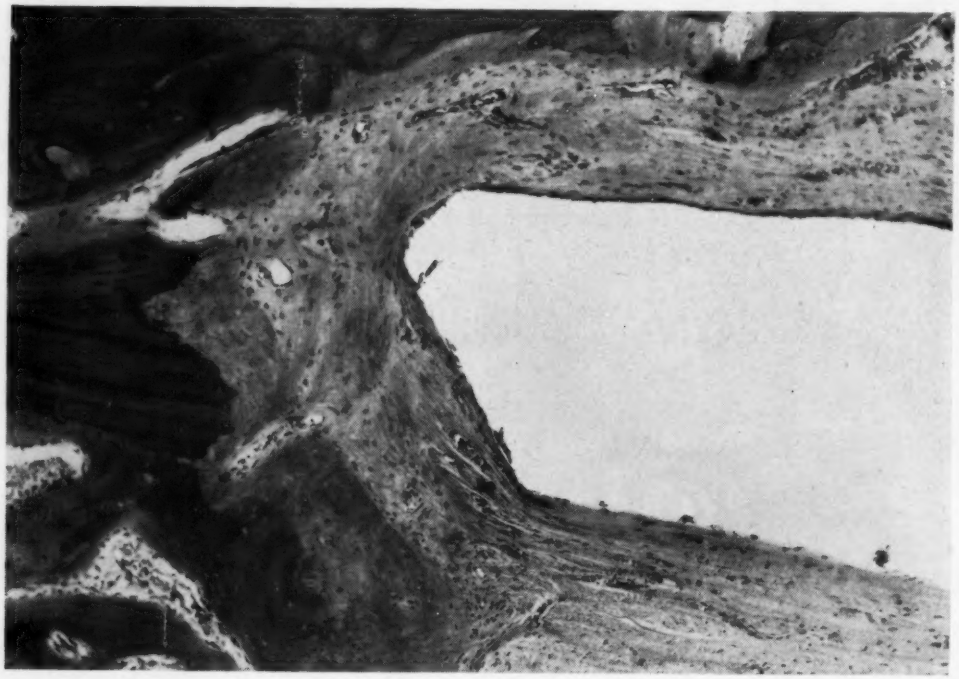

Fig. 7 (Monkey 59).—High-power photomicrograph at the edge of a tantalum implant after 248 days. Note the presence of osteoid tissue adjacent to the old bone. This has been laid down in the outer layers of the dense fibrous tissue membrane surrounding the tantalum.

U-shaped pieces of Lucite were fashioned, with the arms of the "U" 4 to 5 mm. in width and the base a little more than the thickness of the cranium in the monkey, that is, 3 to 4 mm. The Lucite itself was about 1.5 mm. in thickness. These U-shaped pieces of Lucite were dipped in water just below the boiling point at the operating table and bent slightly to conform to the contour of the skull. They were then inserted over the lateral margin of the saw cut, as illustrated in Fig. 1, *B*. The plastic was held in place with two silk sutures. Lucite was inserted in this manner in two monkeys, and the specimens were examined in 172 and 188 days.

There was no gross evidence of inflammatory or foreign body reaction adjacent to this plastic. It was enclosed in a fibrous tissue envelope of about the same thickness as that surrounding tantalum. There was no fluid in the envelope. The surface of the membrane adjacent to the plastic was nonadherent, smooth, and glistening. There was no invasion of the fibrous tissue membrane around the plastic by new bone, either on the periosteal or dural surface. Therefore, no bridging of the artificial gap had resulted.

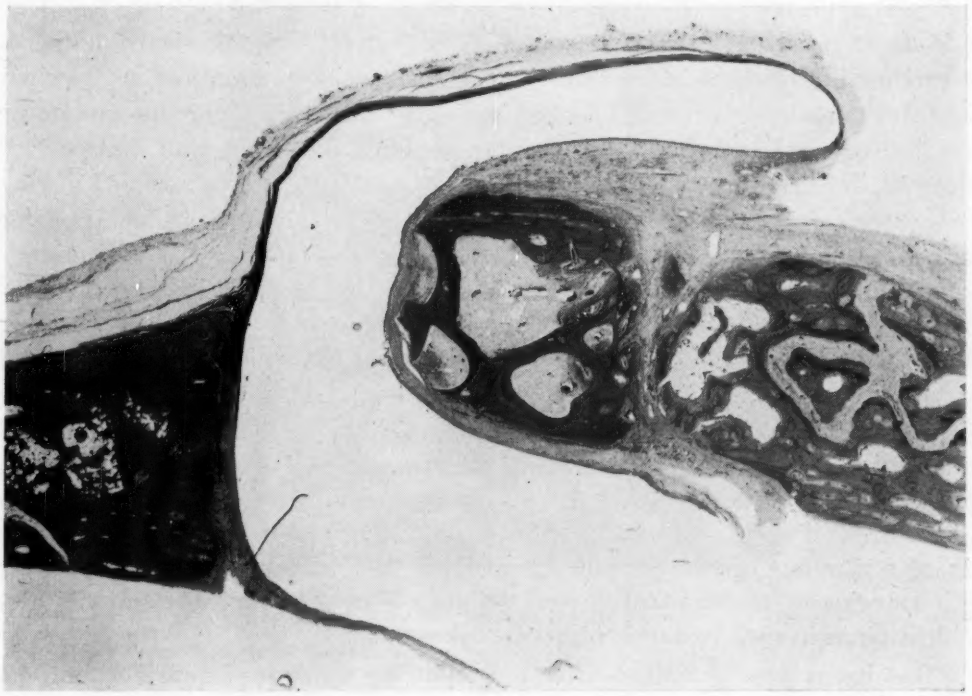

Fig. 8 (Monkey 67).—Low-power photomicrograph (×20) of cross section through cranial saw cut in which a U-shaped piece of methyl methacrylate (Lucite) had been inserted 171 days previously. The fibrous tissue envelope surrounding the plastic is clearly visible. Dense fibrous tissue has invaded a drill hole through the bone on the right. A silk suture had been passed through this drill hole to hold the plastic in place.

Histologic examination of cross sections through the saw cut after removal of the Lucite revealed a well-defined dense fibrous tissue membrane completely enveloping the plastic in the saw cut. The border adjacent to the plastic had a smooth, even surface. There were small blood vessels in the deeper layers of the membrane. There was no evidence of round-cell infiltration and there were no foreign body giant cells adjacent to the plastic. The membrane was about twice as thick between the plastic and the bone as it was over the outer surface of the plastic. Small islands of new bone formation were seen underneath the membrane at the margins of the saw cut, but no attempt had been made to bridge the gap over the surface of the plastic, either on the dural or periosteal aspect (Fig. 8).

Conclusion.—The tolerance of methyl methacrylate by bone and other tissues of the body has been confirmed. No evidence of bony fusion across the plastic

inserted in a saw cut occurred in six months' time. Lucite proved a difficult substance to mold into shapes suitable for this procedure, and it made a palpable swelling beneath the scalp. Because of its rigidity and brittleness, its applicability to placement over the margins of artificial sutures in very young, rapidly growing infants seems doubtful.

Polyethylene (Polythene).—Polyethylene is a pure synthetic plastic substance, a simple polymer of ethylene. It is a tough, thermoplastic resin, slightly cloudy in appearance, which is flexible, resistant to water, and chemically inert. It is light, resistant to temperature changes found within the body, and can be produced easily and inexpensively in flexible tubes and films of any desired size. Its tolerance by the body when buried in the central nervous system or over its surface has been reported by Ingraham, Alexander, and Matson.[26, 30] It can be sterilized by boiling, in which case it retains the shape assumed during the process of boiling, or by immersion in 1:1000 solution of Zephiran for periods of eighteen hours or longer. It cannot be autoclaved. Preliminary experiments with this material seemed to indicate that it would be a particularly suitable substance to introduce into artificial sutures to delay bony healing indefinitely; therefore, it was implanted in cranial saw cuts in seven dogs and six monkeys.

Tubes of pure polyethylene with a wall thickness of 1 mm. were slit longitudinally and inserted over the lateral edge of a saw cut in six monkeys and four dogs, as shown in Fig. 1, *A*. The plastic was held in position with two silk sutures. Polyethylene film, 0.020 in. in thickness, was used over one margin of a saw cut in two puppies (Figs. 1, *C* and 9) and it was placed over both margins of the saw cut in one puppy. The polyethylene film was also held in place in each instance with two silk sutures.

Specimens were examined after the following number of days: 32, 42, 45, 68, 70, 113, 114, 115, 118, 199, 268, 292, and 315. With the exception of one puppy, who suffered a superficial postoperative wound infection, there was no gross evidence of inflammatory or unusual foreign body reaction around the plastic material. The plastic in every case was enveloped by a thin fibrous tissue membrane which was somewhat thicker in specimens older than 100 days than in those examined earlier. In every case the membrane was nonadherent to the plastic, and its surface was smooth and glistening. There were no fluid collections in these fibrous envelopes around the plastic material. The bone edge adjacent to the plastic appeared eburnated and smooth, as shown in Fig. 10. The bone edges adjacent to the plastic were movable to a slight degree, as if a false joint had been formed. In one of the puppies, new bone formation had occurred across the periosteal surface of the film at one end of the saw cut. This was nonadherent to the plastic and had not united with bone on the opposite side, so that there was no actual bony fusion across the artificially created suture. Presumably, this bone grew from periosteum back of the edge of the plastic. There was no new bone formation underneath the plastic where the periosteum had been removed. There was no new bone formation in this case or in any other, on the dural surface.

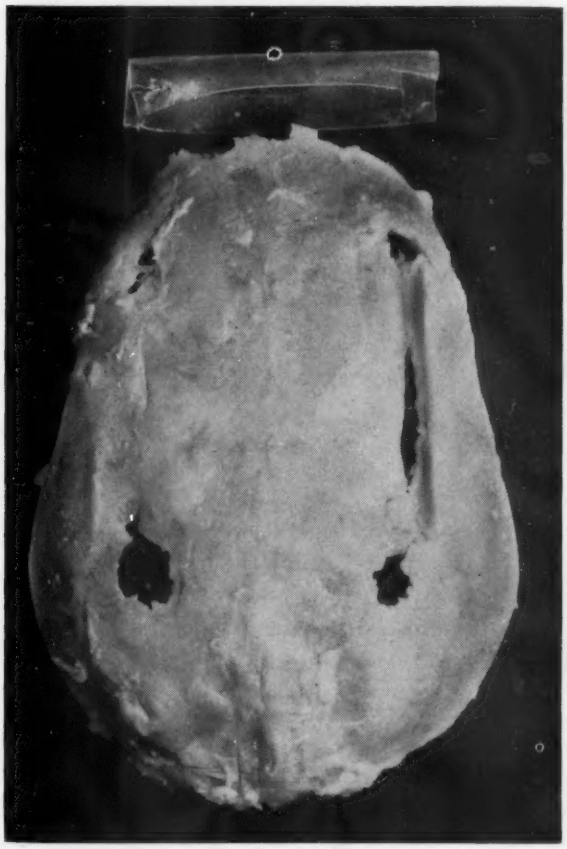

Fig. 9 (Dog 144).—Appearance of calvarium thirty-two days after insertion of polyethylene film in saw cut of a young puppy. On the left the control saw cut has already healed solidly. The piece of film has been removed from the saw cut on the right. There was no attempt at new-bone formation bridging the plastic.

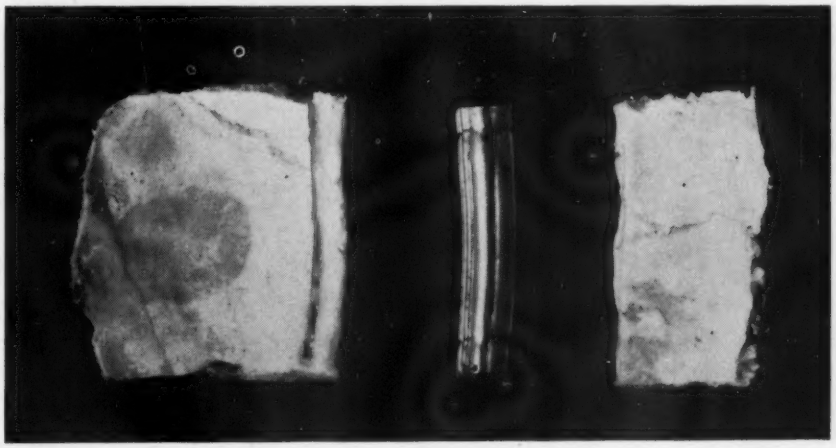

Fig. 10 (Monkey 55).—Segments of cranium 292 days after insertion of polyethylene tube in cranial saw cut. A thin fibrous tissue membrane which completely enveloped the plastic has been removed. Note: (1) On the left the smooth, eburnated edge of the bone margin which had been enclosed by the polyethylene; (2) the smooth, glistening surface of the plastic after being buried 9½ months. There was no new-bone formation in the membrane around the plastic, either adjacent to the edges of the bone or on the pericranial or dural surface.

In one very young puppy, when the cranium was examined after 115 days, marked asymmetry in the growth of the skull was noted. The side where the polyethylene had been inserted in the saw cut was more than 1.5 cm. wider than the opposite half of the cranium at a corresponding point. Specimens of polyethylene removed after periods up to 315 days showed that the plastic had become slightly more translucent and slightly more rigid. However, the material appeared as tough as before, maintained its smooth glistening surface, and was still flexible (Fig. 10).

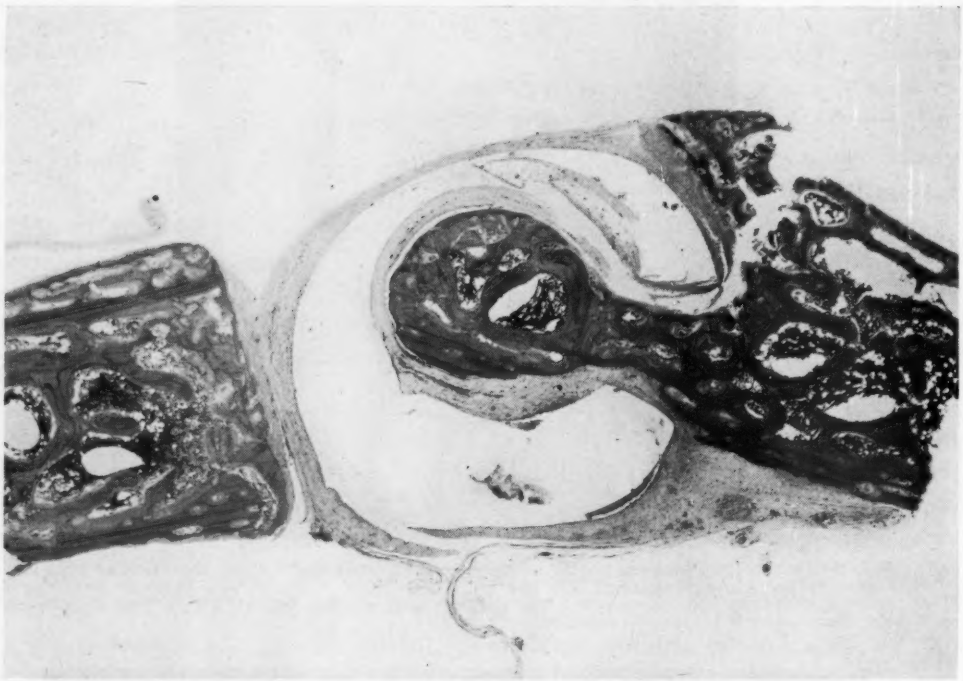

Fig. 11 (Monkey 53).—Low-power photomicrograph (×20) of cross section through the site of implantation of a section of polyethylene tubing over one margin of a saw cut in the parietal bone.

Note: (1) The fibrous tissue envelope completely surrounding the plastic; (2) the smooth, eburnated surface of the bone adjacent to the plastic, resulting in the formation of a false joint.

Histologic examinations of cross sections through the area of the saw cut after removal of the polyethylene were made. These showed that a fibrous tissue membrane enveloped the plastic tubing or film in every case, isolating it from the cut ends of the bone as well as from the dura beneath and the scalp above (Figs. 11 and 13). This fibrous tissue envelope did not contain fluid. It consisted of densely packed, longitudinally arranged fibroblasts. The surface adjacent to the plastic consisted of elongated, flattened cells, having the appearance of serosa. The deeper layers of the membrane contained small blood vessels. The membrane was considerably thicker between the plastic and the bone than on the outer surface of the plastic. The membrane surrounding the fairly thick-walled polyethylene tubing was considerably thicker than that surrounding the thin film. There was no invasion of this membrane ad-

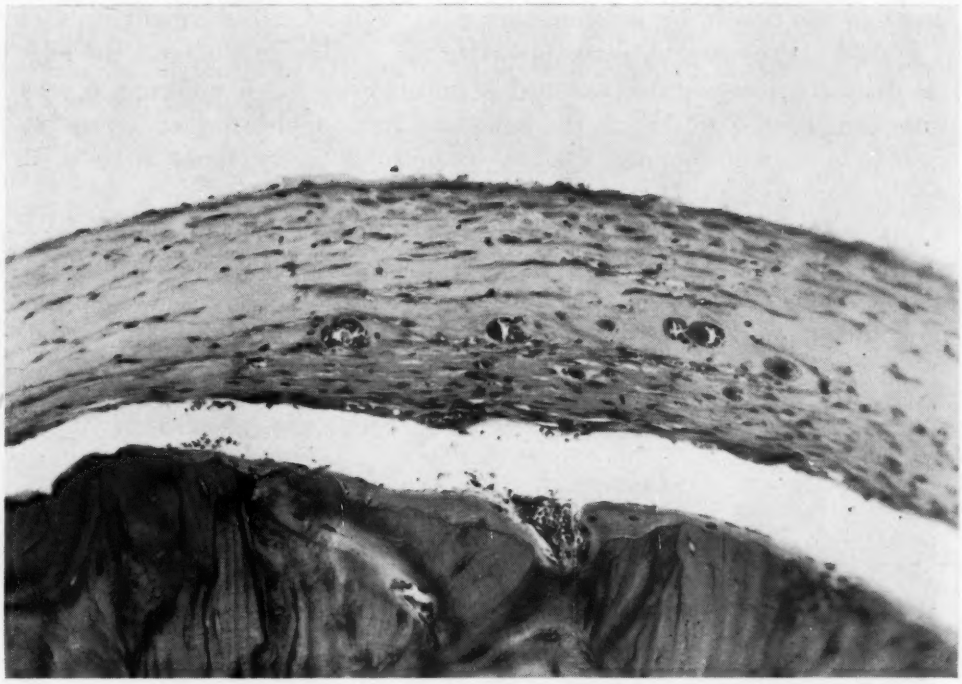

Fig. 12 (Monkey 53).—High-power photomicrograph of the membrane between the cut surface of bone and polyethylene tubing which was removed from the space at the upper margin of the picture. The compact arrangement of the fibrous tissue is evident. The complete lack of inflammatory or foreign body giant cells in the membrane adjacent to the plastic should be noted.

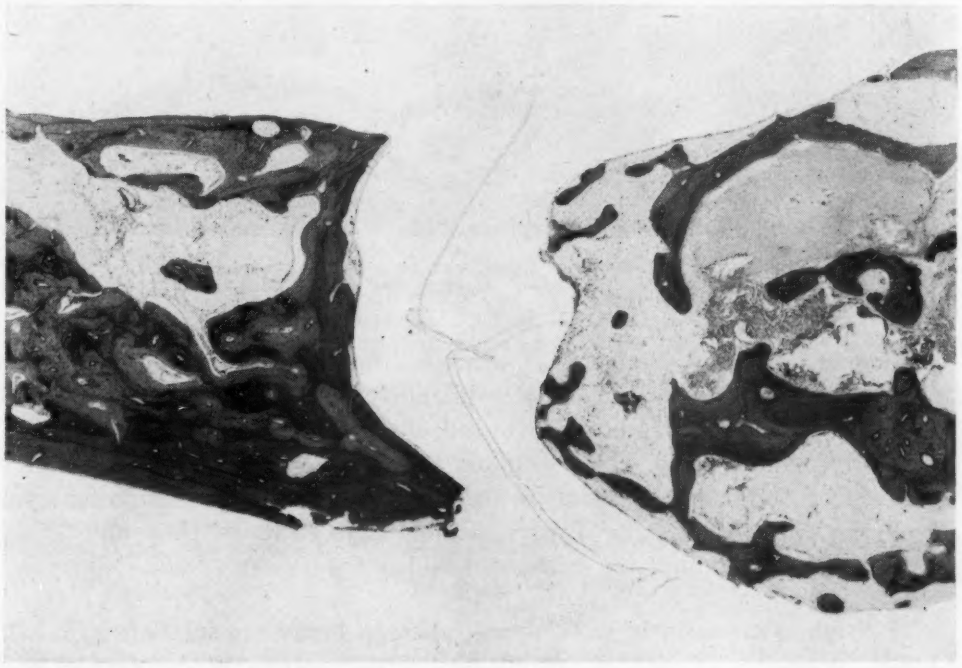

Fig. 13 (Dog 138).—Low-power photomicrograph (×20) of cross section through cranial saw cut in which may be seen a piece of polyethylene film which had been inserted 115 days previously. The thin filmy membrane which has formed over the cut surface of the bone is also visible. The dense fibrous tissue in the marrow at the right is adjacent to remnants of the silk suture passed through the bone at this point.

jacent to the plastic by inflammatory cells or foreign body giant cells (Figs. 12 and 14). The bone edges adjacent to the plastic, particularly the edge enclosed by the plastic tube, assumed a smooth, eburnated appearance; that is, dense bone similar to that of the inner and outer tables formed across the cut edge, in a smooth rounded pattern conforming to the inner surface of the plastic tubing (Figs. 11 and 12).

It is interesting to note that there was considerably more fibrous tissue reaction and cellular infiltration around the silk sutures used to hold the plastic in place than there was around the plastic itself. New bone did not grow into the fibrous envelope which surrounded the plastic, in any instance.

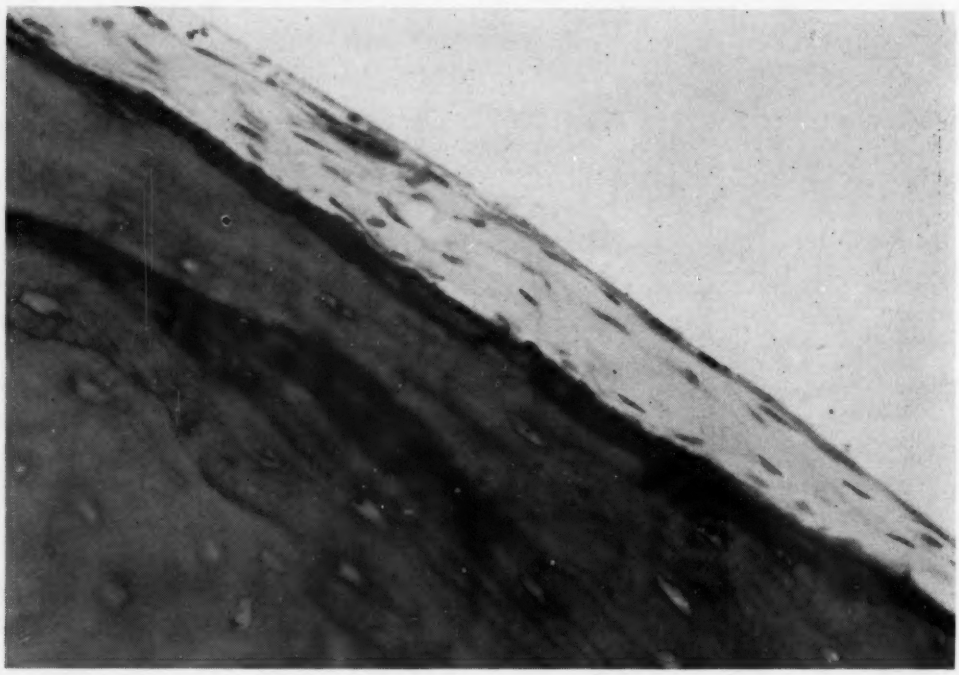

Fig. 14 (Dog 138).—High-power photomicrograph showing the character of the membrane which has formed between the cut surface of bone and a piece of .020 in. thick polyethylene film implanted 115 days previously.

Conclusion.—Polyethylene has proved a foreign substance which, because of its flexibility, transparency, and availability in various sizes of tubing and film, seems particularly adaptable technically to introduction into the gap of artificially made sutures in the cranium in order to inhibit bony healing. Its tolerance by the tissues is equal to that of tantalum and methyl methacrylate; its physical properties make it more suitable than either of these substances.

SUMMARY

1. Craniosynostosis is an obscure inherent mesenchymal defect in which premature closure of cranial sutures produces a variety of deformities of the head and results in mechanical restriction of normal brain growth during infancy.

2. It is particularly important during the first twelve months of life that expansion of the brain be allowed to proceed uninhibited if cerebral deficiency and blindness are to be avoided.

3. Because of the rapid regeneration of bone in this age group it seems desirable to devise a means of creating artificial sutures analogous to those which have fused prematurely and whose closure can be delayed indefinitely or permanently prevented.

4. The interposition of various foreign substances to accomplish this delayed closure of artificial cranial sutures has been investigated in dogs and monkeys. Fibrin film, oxidized cellulose gauze, tantalum, methyl methacrylate, and polyethylene were studied.

5. Of this group, the synthetic plastic, polyethylene, possesses the most desirable physical properties. It can be obtained and used as flexible hollow tubing or as a thin film. The latter form is more suitable for the application considered in this report. Polyethylene is well tolerated by bone and other body tissues. In periods up to nine and one-half months it remained physically inert in the body.

6. Preliminary observations of clinical trials of this method in six infants with craniosynostosis have been favorable. These will be reported in detail at a later date.

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CYST FORMATION AT THE OPERATIVE SITE FOLLOWING CEREBELLAR OPERATIONS (PSEUDOMENINGOCELE)

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AN OCCASIONAL troublesome complication following operations for lesions in the posterior cranial fossa is the collection of a large amount of fluid under the occipital muscles at the operative site. In the last ten years, this complication has been present in fourteen patients who have had operations in the posterior fossa at the University of Minnesota Hospitals. The seriousness of this complication is illustrated by the fact that it caused a considerable increase in the period of hospitalization in most of these cases, and in three of them a second operation was performed because of the protrusion at the operative site. One patient died as a result of this complication. The results obtained by the use of a simple pressure dressing over the protruding area indicate that these protrusions may be treated quite successfully by this method.

A review of the literature from 1916 to 1946 reveals very few references to cyst formation at the operative site following intracranial surgery, but the fact that this complication has been occasionally mentioned indicates that it has been seen and recognized by others. Schloffer[1] in 1923 reported a case of meningioma of the right parietal area which was completely removed in two stages. Following removal of the tumor, there was a marked increase in the cerebrospinal fluid pressure which was relieved by periodic lumbar punctures with drainage of from 20 to 125 c.c. of cerebrospinal fluid during each puncture. There was marked protrusion of the scalp over the operative site which was reduced with each lumbar puncture. This protrusion persisted for a period of eight months following operation, but gradually disappeared. One year following the removal of the tumor this patient was perfectly well. The protrusion in this case did not follow a cerebellar exploration, as was true in our cases, but otherwise it was similar. Cushing apparently has made no direct mention of this problem, but that he was aware that such cyst formation followed cerebellar operations is evidenced by comments in at least two of his papers dealing with cerebellar tumors.[2, 3] In discussing the postoperative course of individual cases in these papers he mentioned several times that there was no protrusion at the operative site. Bucy[5] has reported that Cushing applied the term "pseudomeningocele" to this condition. Since the protrusion is apparently due to a collection of cerebrospinal fluid in a cystic cavity under the suboccipital muscles, this term seems appropriate for the condition and we have continued its use.

Pseudomeningoceles were present in fourteen of our patients who had cerebellar operations within the last ten years. In the group there were six cases

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of cystic astrocytoma of the cerebellum, one hemangioma of the cerebellum, one pinealoma, and two cases of obstruction of the aqueduct of undetermined etiology. In seven of these cases a curved incision extending from one mastoid process to the other was used; in four cases a straight midline incision was used; in two the incision was of the hockey stick type for unilateral cerebellar exploration; and in one case the type of incision was unknown. The dura was closed in six cases; in two cases it was left open. It appears then, that pseudomeningoceles may occur with any type of incision, and that closure of the dura will not prevent their occurrence.

Fig. 1.—A typical pseudomeningocele which occurred following a right suboccipital craniotomy for removal of a meningioma from the posterior fossa.

Eight patients in this group were operated upon before 1944 when pressure dressings were first used to treat pseudomeningoceles. Five of these eight patients were treated either by repeated aspirations of the cystic cavity or by lumbar puncture with drainage of cerebrospinal fluid to decompress the pseudomeningocele. In three of the cases no aspirations were performed. In one case a cerebrospinal fluid fistula developed as a result of the pseudomeningocele and the patient died of meningitis secondary to this fistula. Three of these eight patients were reoperated upon because the nature of the pseudomeningocele was not recognized, and it was thought that they had increased intracranial pressure due to recurrence of the tumor. In each of these three cases a cystic cavity filled with clear colorless fluid was found under the occipital muscles, and there was no recurrence of the tumor. The cysts were lined by thick, glistening, white membranes. Biopsy of the cyst walls revealed that they were composed of collagenous connective tissue. No communication was found at operation be-

tween the cyst cavity and the subarachnoid space, but that such a communication does exist in these cases is shown by the fact that the cyst can be completely collapsed by aspiration of cerebrospinal fluid by the lumbar route. In addition, the fluid in the pseudomeningocele is chemically identical with the cerebrospinal fluid removed at the same time from the lumbar subarachnoid space.

Six patients with pseudomeningocele have been seen since 1944. All of these have been treated by the application of a pressure dressing over the pseudomeningocele. In five of these patients, the pseudomeningocele quickly disappeared following the application of the pressure dressing. In one case the pressure dressing failed to cure the condition, but this failure was probably due to the fact

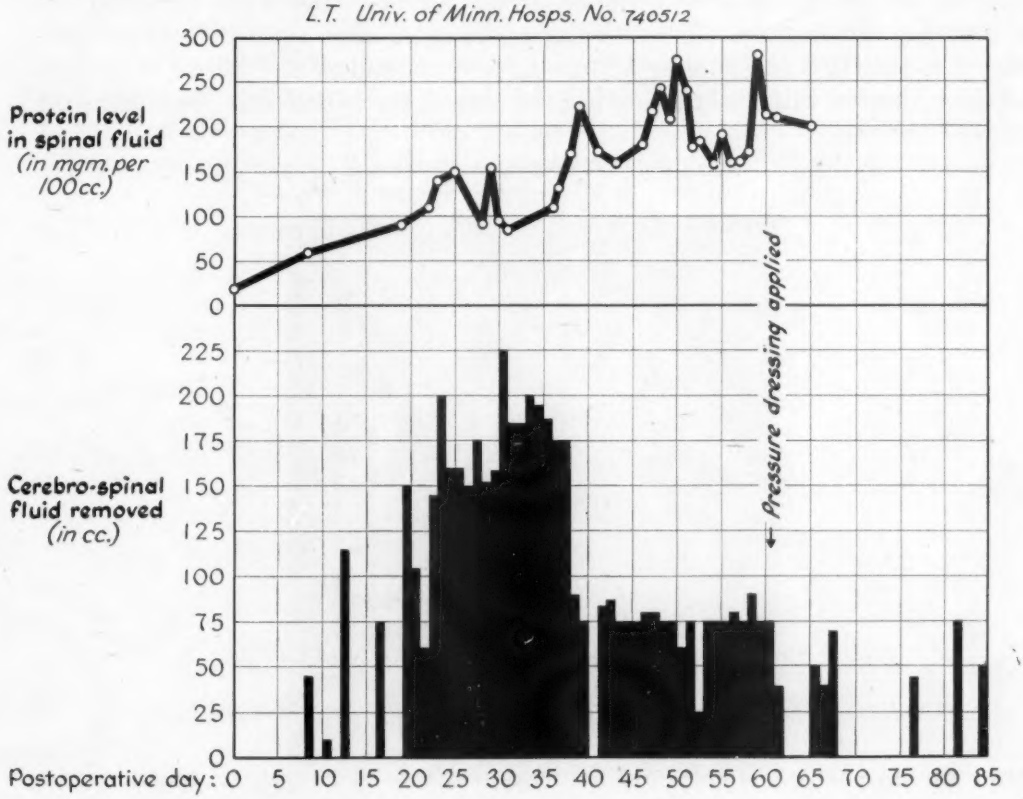

Fig. 2.—Graph showing amounts of fluid removed to decompress the pseudomeningocele before and after the application of pressure dressings.

that the tumor could not be completely resected. The patient continued to have increased intracranial pressure following the operation. The first two patients of this group who were treated by the application of a pressure dressing were first treated by repeated aspirations of cerebrospinal fluid. Figs. 2 and 3 are graphs summarizing the treatment of these two patients by aspiration of cerebrospinal fluid. These graphs also illustrate how effectively the pseudomeningoceles were treated by the use of a pressure dressing. In the first of these two cases (Fig. 2) there was so little accumulation of fluid following the application of a pressure dressing that subsequent aspirations were done only seven times.

We realize now, although we did not at the time this patient was being treated, that even these aspirations were unnecessary. In the second case (Fig. 3) there was accumulation of fluid in the cystic cavity only twice after the pressure dressing was applied, and these two recurrences of the pseudomeningocele occurred when the child pulled off the pressure dressing.

Pseudomeningoceles must be due either to an increased secretion or to diminished absorption of cerebrospinal fluid, or possibly to both of these factors. Flexner[5] has shown that the formation of cerebrospinal fluid is a linear function of the difference between the effective hydrostatic pressures in the capillaries of the choroid plexus and in the ventricles. Weed[6, 7] has demonstrated that an increase in the hydrostatic pressure of the cerebrospinal fluid increased the absorption of the fluid. In cerebellar operations, large skull defects are produced so that it is possible for a large amount of fluid to accumulate at the site of the operation without bringing back to normal the hydrostatic pressure in the

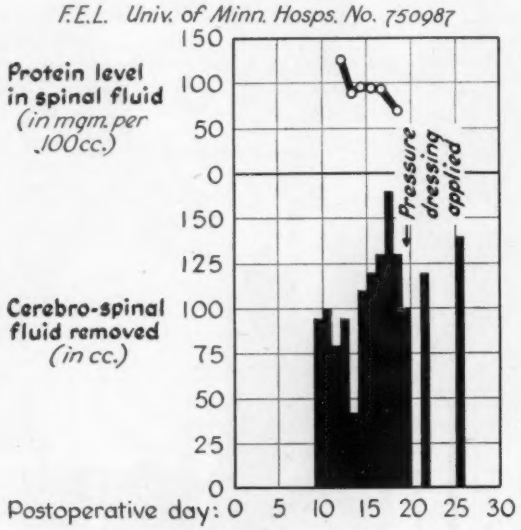

Fig. 3.—Graph showing amounts of fluid removed from pseudomeningocele.

craniospinal cavity. Thus, fluid accumulates to form a cyst, or pseudomeningocele, until the area of the wound becomes tense and the hydrostatic pressure of the cerebrospinal fluid is raised to a level at which normal relationships between absorption and secretion are resumed, but because the cyst causes stretching of the soft tissues the pseudomeningocele may become progressively larger. Application of a pressure dressing over the pseudomeningocele arrests the process because its support of the soft tissues over the skull defect causes an increase in hydrostatic pressure of the cerebrospinal fluid without protrusion of the operative site.

The most satisfactory method for applying moderate pressure to the suboccipital area is by the use of an elastic bandage as illustrated in Fig. 4. Some patients complain of mild headache for the first twenty-four to forty-eight hours after the bandage is applied, but there is no other discomfort. The length

of time that the dressing is needed varies from patient to patient. The procedure which we have used and found to be satisfactory is to apply the dressing for five days and then to discontinue its use unless there is recurrence of the pseudomeningocele. If there is recurrence, the dressing is used for an additional two or three days. Again its use is discontinued to discover whether the tendency to cyst formation has been eliminated. This procedure is repeated until the tendency to develop a pseudomeningocele has completely disappeared. Frequently it is found that the pseudomeningocele recurs only at certain times during the day, so that a compression dressing is necessary only at these times. It has been found unnecessary for the patient to remain in the hospital if tendency to form pseudomeningocele is prolonged, for a member of the patient's family can be easily taught to apply the pressure dressing. Only a moderate amount of pressure is needed to control the protrusion so that the bandage should be applied snugly, but not so tightly that there is danger of pressure necrosis to the underlying skin. However, if there is any doubt about the condition of the skin, the dressing may be removed daily and immediately replaced.

Fig. 4.—A patient with a pseudomeningocele with pressure dressing in place.

Following is the case report of a patient with a pseudomeningocele. This case illustrates the ease with which this complication may be treated with pressure dressings.

B. R., a 33-year-old white woman, was operated upon Aug. 6, 1945. A straight midline cerebellar incision was used and a large meningioma was found, having its origin from the inferior surface of the tentorium on the right side of the posterior fossa. The tumor was completely removed and the dura, subcutaneous tissues, and skin were closed in layers. The patient's immediate postoperative course was uneventful, and she was discharged from the hospital on the fourth postoperative day.

On the eighth postoperative day, the patient noticed some protrusion at the operative site. This increased until the eighteenth postoperative day when she returned to the outpatient clinic

because she had become alarmed about this swelling. The area was tense and protruded about 3 cm. An elastic bandage was applied over the protruding area and was left in place for twenty-four hours. At the end of this twenty-four hour period the operative area was soft and flat. The pressure dressing was then removed, but within three hours the swelling had recurred. The bandage was again applied and left in place for three more days. At the end of this time, the dressing was again removed, but the pseudomeningocele recurred within a few hours. The dressing was then reapplied and left in place for another three days. It was then removed. This period of compression apparently was sufficient, for there was no further protrusion at the operative site. The patient was followed in the outpatient service for one year after operation and remained completely free of symptoms.

SUMMARY

Pseudomeningocele or cyst formation at the operative site following cerebellar operations has received little mention in the literature, but apparently is frequently encountered by neurosurgeons.

We have encountered this complication fourteen times during a ten-year period.

A simple but efficient method for the elimination of meningoceles has been found in the application of a pressure dressing.

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FOREIGN BODY LOCALIZATION IN THE SOFT PARTS

A Simple Method Requiring no Especial Training or Equipment

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THE search for and removal of foreign bodies in the soft parts can be one of the most difficult and exasperating of surgical problems. Methods described to assist in the procedure have become numerous and complex and often require elaborate equipment and specialized training which is not available in the ordinary hospital. A procedure is here described which is simple and satisfactory and requires no especial equipment or training.

Reed and Black,[1] in a review of the literature in 1938, classified the methods of foreign body localization under ten different principles of which 110 methods were reported. Since then the literature has contained many references to refinements in these methods, and the introduction of at least one new principle.[2]

The method here described is to us the simplest and most satisfactory and is the one least mentioned in the literature. It consists in the insertion under fluoroscopic control of two long slender needles at right angles into the tissues at a distance from the proposed incision so that they cross in the approximate location of the foreign body. An anteroposterior and lateral x-ray film is then exposed and with the needles still in place the field is draped and the area explored at operation. The foreign body can usually be quickly found because of the ease in localizing it in relation to the fixed point of the two crossed needles. If difficulty is still experienced, the needles are withdrawn and two more inserted in the same manner so that they cross again at the approximate location of the foreign body. The wound is then covered, anteroposterior and lateral x-ray views again taken and, with the needles acting as fixed points for localization, we have never failed to find even the tiniest of foreign bodies.

Most of the methods used in foreign body localization rely upon skin markings made with the aid of x-ray examination. The difficulty with this method is that the foreign body may shift in relation to the skin mark if the patient is not in exactly the same position on the operating table as under the x-ray screen. Furthermore, during the course of operation both foreign body and the skin marks shift as tissues are retracted.

The method described eliminates this error, for the two crossed needles anchor the tissues, and their relation to the foreign body does not change. It is only when the needles become loosened in the tissues during the dissection and no longer act as fixed points that they may need to be withdrawn and reinserted for a second x-ray exposure.

Although the method has been previously described,[3, 4] we developed the technique independently in military service and have subsequently used it in civilian practice. The following case report in which a thoracentesis needle

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broke off flush with the inner margin of a rib is an example of an extremely difficult foreign body to find, in which the method was used with excellent results.

CASE REPORT

T. P. (No. B 91370), a 15-year-old Negro girl, was hospitalized elsewhere when a lumbar puncture needle inserted into the right thorax for thoracentesis broke off at its hub. Immediate exploration through a 3 cm. incision was unsuccessful and the patient was brought to us. Fluoroscopy showed an 8 cm. needle entirely within the right thorax except for its distal end, which was in the chest wall between the eighth and ninth ribs at the posterior axillary line (Fig. 1). Its most superficial end appeared to be on a level with the mid-portion of the rib (Fig. 1, *c*). Two long 22 gauge needles were thrust through the soft tissues of the chest wall starting them about 3 cm. from the skin incision so that the needles crossed in approximately the location of the needle (Fig. 1, *A*, *B*, and *C*). This was done under the fluoroscope. Using the point where the needles crossed as the fixed

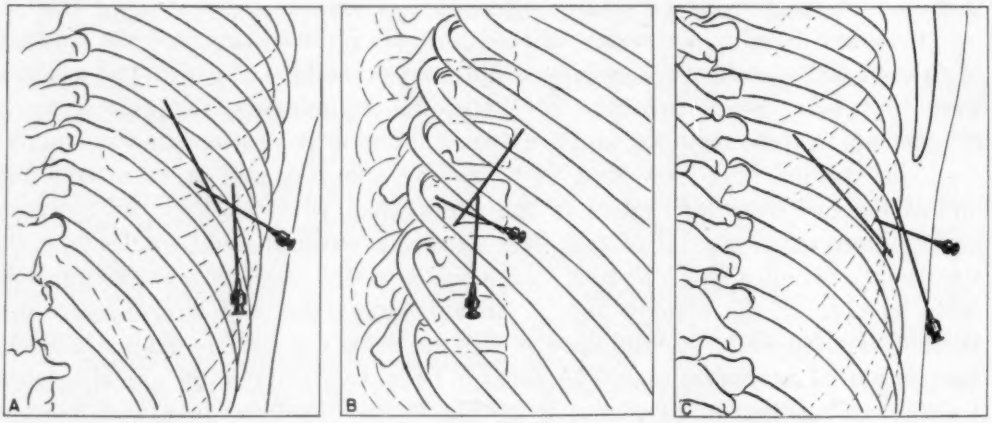

Fig. 1.—Drawings made from tracings of anteroposterior, lateral, and oblique x-ray films after two slender needles had been inserted through the soft tissues of the chest wall so that they crossed in approximately the same position as the end of the broken needle. *A*, anteroposterior; *B*, lateral; *C*, oblique.

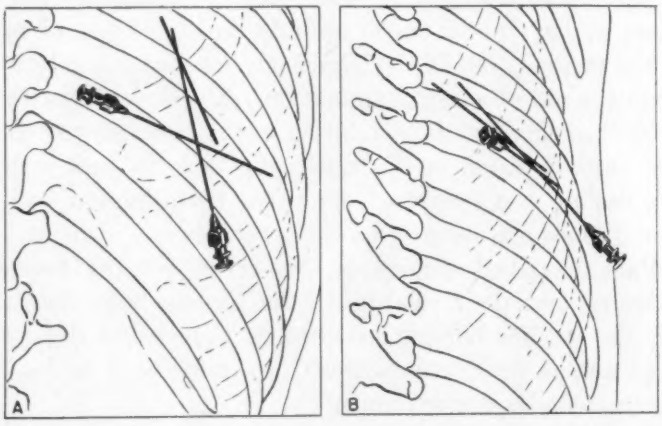

Fig. 2.—Drawings of the anteroposterior and oblique x-ray views made on the operating table after the first set of localizing needles had been loosened and a second set had been inserted through the intercostal space so that they crossed at the approximate location of the broken needle. The end of the broken needle was immediately located by searching along the horizontal of the two localizing needles 1 cm. lateral and 2 mm. above the fixed point found by the crossing of the two needles.

point it was seen that the end of the foreign body was located about 1 cm. caudad, 1 cm. posterior, and ½ cm. deeper than the fixed point. Exploration in this area failed to locate the foreign body quickly and the needles became slightly loosened during the procedure. These needles were withdrawn and two lumbar puncture needles were inserted starting at a distance of some 4 cm. from the wound and inserting the needles into the thoracic cavity crossing them through the interspace at the point where the foreign body was thought to be. Study of anteroposterior and oblique x-ray films (Fig. 2, *A* and *B*) showed that these needles crossed at exactly the same level as the end of the foreign body and that the end of the foreign body was located along the horizontal of the two needles 1 cm. lateral to the crossing and about 2 mm. cephalad. Exploration at this point immediately revealed the broken end of the needle with only about 2 mm. of its length superficial to the pleura. When the needle was withdrawn a little air sucked into the pleural cavity. This was quickly stopped by suturing the muscles over the opening. The previous wound in the skin and subcutaneous tissues was then excised and closed and the patient allowed to return to the original hospital.

SUMMARY

A simple and satisfactory method of localizing foreign bodies in the soft parts, without the need of elaborate equipment or an especially trained staff, is presented.

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SUBCUTANEOUS EMPHYSEMA FOLLOWING CHEST TRAUMA

Analysis of Twenty Cases

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SUBCUTANEOUS emphysema following thoracic trauma is not infrequent and is often seen after blunt chest injuries as well as after penetrating wounds. In the majority of instances active therapy directed toward controlling the spread of emphysema is unnecessary as the process is a self-limited one and subsides spontaneously. There are cases, however, where the emphysema is progressive, respiration becomes much embarrassed, and the circulation is interfered with. Such cases, unchecked, may result in death. An understanding of the pathologic physiology involved is necessary to treat this condition adequately. The tempo of events may be very rapid or last over a period of days. One must realize that other important lesions may be present in various combinations and that the emphysema may be but one phase of a complicated clinical picture.

There are two pathways by which air will reach the subcutaneous tissues from the lungs. Each involves a different mechanism and depends on a different type of pulmonic injury. Nevertheless, one, or the other, or both mechanisms may operate in any one or several cases.

Mechanism 1.—With rib fractures, the parietal pleura, visceral pleura, and underlying lung may be lacerated. Air will pass from the injured lung into subcutaneously lacerated soft tissues via the pleural space with or without an x-ray-apparent pneumothorax in the early phases of the condition. Later x-ray examination may reveal a small pneumothorax or pleural or pulmonary changes not manifested early. In some instances a minimal or partial pneumothorax will be seen early. In expiration, air escapes into the pleural space; with inspiration, it is forced into the soft tissues by the expanding lung, thereby causing and extending the subcutaneous emphysema. This may continue until an obvious pneumothorax forms which will compress the lung and interrupt the further escape of air from it, or until the laceration in the lung becomes sealed over. With a penetrating injury, air may also be sucked into the thoracic parieties from the exterior. This explanation was presented by Stenbuck[1] in 1937.

That air can get from the lung to the subcutaneous tissues without connecting adhesions or x-ray-determined pneumothorax after injury is suggested by the following:

In five of our penetrating thoracic injuries there was x-ray-demonstrable pneumothorax in none, extensive subcutaneous emphysema in three, moderate emphysema in one, and slight emphysema in one.

It is unlikely that the lung was uninvolved in all five cases or that the exterior was the sole source of emphysema by the very nature of the injuries,

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that is, three stab wounds, one transfixion on a spike, and a nail penetration. Furthermore, in one case, pulmonary infiltration and pleural thickening were demonstrable by x-ray examination, indicating that the lung had been injured.

In six cases of unilateral rib fractures, in nonpenetrating injuries, four left and two right, the emphysema was present essentially on the side of the fractures. In four cases, pneumothorax was noted on the same side; in three cases there was homolateral pleuritis or pleural fluid; and in two cases, pulmonary infiltration. In one case of multiple right and one left rib fracture there was extensive right subcutaneous emphysema, right pneumothorax, pleuritis, and diminished aeration of the right lower lung. Such correlation of phenomena strongly suggests the local mechanism just elucidated.

Mechanism 2.—This explanation has been given a number of times in the several articles on the subject by Macklin,[2] and Macklin and Macklin.[3, 4] The demonstrations and data presented were very convincing and reference is made to their publications for details. In essence, as a result of a variety of factors, that is, external trauma with or without rib fracture, overinflation of the lungs as a result of inhalation anesthesia, resuscitative measures, atelectasis for one cause or another with compensatory emphysema, straining, and a congenital predisposition on the part of the subject, rupture of overdistended alveoli take place. These are usually multiple and may be demonstrable by microscopic and special techniques elaborated by Macklin and Macklin, occasionally by ordinary observation. Air escapes from the alveoli into the perivascular sheaths of capillaries and larger blood vessels. As a result of inspiration and expiration further expulsion of air takes place.

Air bubbles coalesce and are pushed along throughout the lung substance down toward the lung root into the mediastinum. They may infiltrate across the other side into the opposite lung root. The mediastinal pressure builds up. Occasionally the air remains trapped and causes marked cyanosis and dyspnea because of the avascularity of the lung involved, and the pressure on the great mediastinal veins impeding the return flow of blood to the heart. Death may result. There are three main routes of egress from the mediastinum; the subcutaneous tissues of the neck, the pleural cavities, and the retroperitoneal space. Air may escape into any one or more of these, and will cause subcutaneous emphysema, pneumothorax, or retroperitoneal emphysema. Subcutaneous emphysema may become generalized. Even when air enters one of the escape routes, pressure symptoms may still occur and constitute a threat to life.

In eight of our cases collected, this mechanism alone, or possibly in conjunction with the first described, may have operated. The symptomatology and course of events are best explained on the basis of this concept. The following two cases are presented in some detail as they appear to illustrate the second mechanism described.

CASE REPORTS

CASE 1 (No. 117493).—W. G. was admitted Oct. 22, 1946 and discharged Nov. 12, 1946. This patient was a 62-year-old white man who fell off a steam-heat radiator and struck the right side of the chest against it. He then complained of pain in the right side of

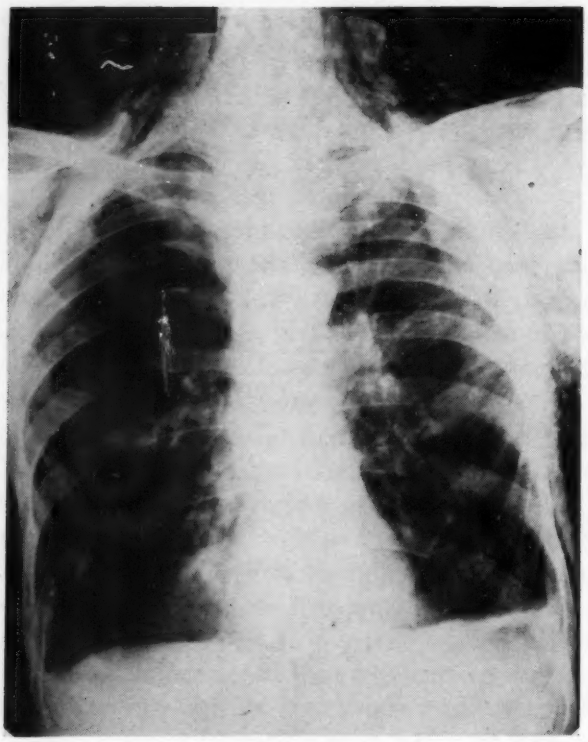

Fig. 1 (Case 1).—X-ray view taken Oct. 22, 1946, showing marked subcutaneous emphysema of thoracic wall and cervical areas.

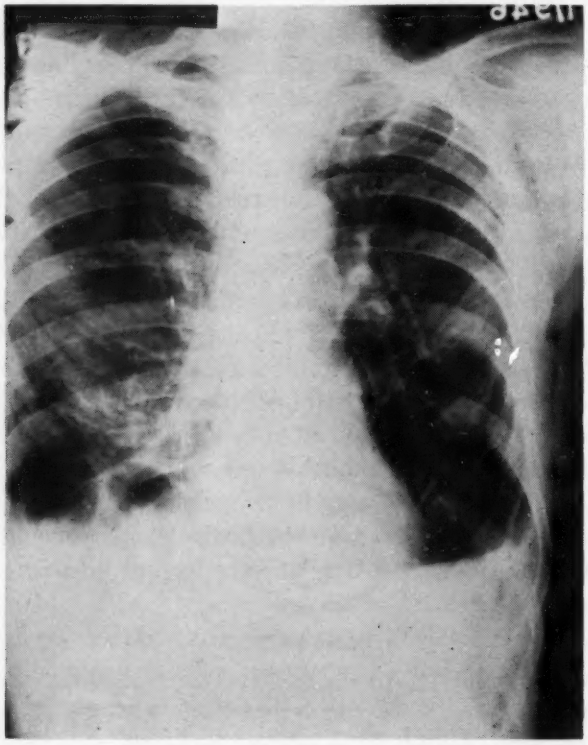

Fig. 2 (Case 1).—X-ray view taken Oct. 29, 1946, showing diminished subcutaneous emphysema, partial pneumothorax of the right lower lung, and pleural thickening of both bases.

the chest and some difficulty in breathing. His physician noticed increasing crepitation of the chest wall over a two-day period and referred him for hospitalization.

Physical examination on admission Oct. 22, 1946, revealed moderate respiratory distress. Subcutaneous emphysema was present over the entire chest, subcostal margins, and flanks. There was no skin laceration. The remainder of the examination was essentially irrelevant. Laboratory data: Hemoglobin was 13 Gm.; white blood cells, 9000; blood Wassermann, negative; x-ray examination on Oct. 23, 1946, showed a marked degree of subcutaneous emphysema in the thoracic wall and in the cervical regions of both sides, particularly on the *left.* There were complete fractures of the right seventh, eighth, ninth, and tenth ribs in the axillary region (Fig. 1).

Course in the Hospital.—The chest was strapped over the area of the rib fractures but the strapping was poorly tolerated and had to be discontinued. For the next few days the emphysema continued to extend until on Oct. 27, 1946, it became universal, involving the entire body from the scalp to the ankles. The eyelids were puffed up and closed. Cyanosis, dyspnea, and orthopnea developed. At this time there was no demonstrable pneumothorax on either side, no mediastinal shift, and no significant pulmonary changes. It was apparent that active treatment was necessary and further expectant therapy dangerous to the patient's life. Accordingly, under novocain infiltration, a small horizontal one-inch, suprasternal incision was made. The incision was carried down through all the layers of fascia and subcutaneous tissue to the trachea. The finger was then bluntly introduced into the superior mediastinum behind the sternum. Air bubbled out through the incision continuously during the operation. A Penrose drain was introduced into the space which had been opened. Within ten minutes the dyspnea and orthopnea were relieved. Recovery thereafter was continuous and within one week there was almost complete disappearance of the subcutaneous emphysema. For the first ten days a low-grade febrile course persisted and penicillin was administered throughout this time. Chest x-ray view on Oct. 29, 1946, showed a considerable degree of subcutaneous emphysema of the entire thorax and cervical region (less than on the previous film), a partial pneumothorax at the right lower lung, and pleural thickening of both bases (Fig. 2).

CASE 2 (No. 118268).—J. S. was admitted Dec. 17, 1946, and discharged Jan. 10, 1947. This patient was a 57-year-old man who was attacked by two bandits, and sustained ten fractured ribs about equally distributed on either side.

Examination on admission disclosed subcutaneous emphysema of the entire chest wall, front and back, extending up to the face and down to the iliac crests. It was most marked posteriorly. Pain was present on inspiration but dyspnea and cyanosis were absent.

X-ray examination on admission showed marked subcutaneous emphysema of the chest wall, cervical area, and the *mediastinum* (Figs. 3 and 4). There were multiple bilateral rib fractures with slight displacement of some fragments. There was partial atelectasis or hemorrhage in the right lower lung.

Course in the Hospital.—The chest was strapped and the patient treated expectantly. For two days there was extension of the emphysema and then it began to subside. Recovery was uneventful. X-ray examination of the chest on Jan. 2, 1947, revealed minimal degree of pulmonary infiltration at the right base. The right diaphragm was elevated. X-ray examination on Jan. 8, 1947, was essentially negative.

Table I is a summary of the pertinent data in twenty cases of subcutaneous emphysema following chest injury encountered at the Coney Island Hospital in the past nine years.

Five were penetrating chest wounds. X-ray-demonstrable pneumothorax was not present in any. There was a small pleural effusion in one case, and slight pulmonary infiltration in the right upper lobe with pleural thickening in another.

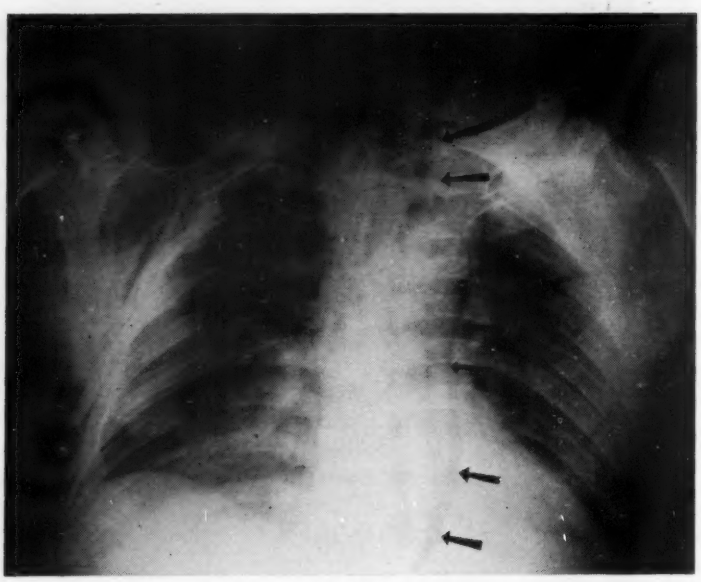

Fig. 3 (Case 2).—X-ray view showing subcutaneous emphysema of chest wall, cervical area, and mediastinum.

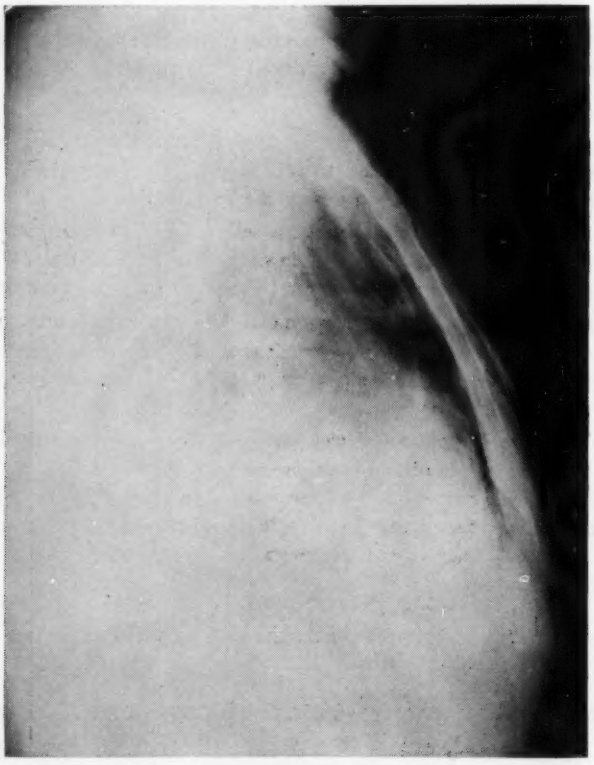

Fig. 4 (Case 2).—Lateral X-ray view showing subcutaneous and mediastinal emphysema.

TABLE I. SUMMARY OF DATA IN TWENTY CASES OF SUBCUTANEOUS EMPHYSEMA FOLLOWING CHEST TRAUMA

CASE NO., AGE, SEX	INJURY	EMPHYSEMA (SITE, EXTENT, COURSE)	PNEUMOTHORAX (EARLY, LATE)	PLEURAL FLUID, PLEURITIS	PULMONARY CHANGES (EARLY, LATE)	RESPIRATORY EMBARRASSMENT	COMMENT
1. C. P. (82221), 26, ♂	Stab wound of 4th right interspace posteriorly	Marked over right posterior chest; uneventful recovery in 5 days	0	Small amount of fluid	0	0	
2. F. W. (88332) 28, ♂	Stab wound of 2nd left interspace parasternally	Marked on left anterior chest from clavicle down; spread to face and subsided spontaneously	0	0	0	0	
3. C. W. (81435), 29, ♀	Stab wound of 4th left interspace of anterior axillary line	Moderate over anterior left chest, supraclavicular area and left posterior chest wall; uneventful recovery in several days	0	0	0	0	
4. N. A. (65278), 17, ♂	Nail laceration of left axillary area, 3rd interspace	Relatively slight, in left axillary area; uneventful recovery	0	0	0	0	
5. F. G. (64857), 13, ♂	Fell on spike; laceration of right anterior and posterior chest	About laceration sites, front and back of chest; no spread, uneventful recovery in 9 days	0	Slight right pleural thickening	Alveolar hemorrhage of right upper lung, early; slight pulmonary infiltration of right upper lung, late	0	These first 5 cases were all penetrating chest injuries
6. S. C. (78590), 67, ♂	Struck by train; multiple left rib fractures, fractured skull	Over left lateral aspect of chest	Left (small) early	Small amount (early blood?)	0	+	Patient died several hours after admission in coma; dyspnea of cerebral origin, death due to head injury
7. D. J. (102859), 76, ♂	Hit by an auto; fracture of left clavicle and right scapula, laceration of scalp, shock, B.P. 85/50, no rib fracture	Extensive over both sides of neck and in right axillary region; spread to abdomen and gluteal area	Right (early)	0	0	++	Death in 24 hr. with marked respiratory embarrassment; while death may have been due to shock and head trauma, the pulmonary complication may have played a large part, decompression of the mediastinum might have relieved the respiratory distress

TABLE I—CONT'D

CASE NO., AGE, SEX	INJURY	EMPHYSEMA (SITE, EXTENT, COURSE)	PNEUMOTHORAX (EARLY, LATE)	PLEURAL FLUID, PLEURITIS	PULMONARY CHANGES (EARLY, LATE)	RESPIRATORY EMBARRASSMENT	COMMENT
8. B. R. (101878), 45, ♂	Struck by car; unconscious, shock, head trauma, multiple bilateral rib fractures	Moderate of left side of chest	0	+ Moderate amount	Some pulmonary infiltration (hemorrhage?)	Developed on day after admission +	Death 24 hr. after admission; respiratory difficulty became marked on day after admission and pulse markedly irregular; while death was probably due to shock and head trauma, the possibility of increased mediastinal pressure is present
9. C. H. (64507), 33, ♂	Struck by car; unconscious, head trauma, right rib fracture	Extensive over anterior and posterior chest, neck, down to below mid-trunk; progressive for 24 hr. down to inguinal areas, then subsidence and rapid convalescence	Possible right (minimal and early)	0	Bilateral emphysema	0	Emphysema spread through mediastinal route (most probable); spontaneous recovery
10. A. S. (61537), 49, ♂	Struck by auto; multiple left rib fractures, laceration of left chest, contusion of left kidney	Entire left chest wall and neck involved; subsided after several days	+ Late left	Pleural thickening and fluid, left lower lung, late	Clouding lower ½ left lung (hemorrhage?) early	0	
11. A. G. (114085), 48, ♂	Struck by auto; fracture 5th & 6th left ribs, linear skull fracture	Anterior chest wall up to clavicle, subsided spontaneously	++1 Left early	+ Left (late)	Collapse of left lung (early)	0	Spontaneous subsidence; early moderate pneumothorax and lung collapse may have caused early stopping of emphysema
12. P. M. (64464), 38, ♂	Struck by auto; unconscious, multiple right rib fractures	Moderate right thoracic wall and right cervical region; subsided spontaneously after several days	+ Right early (minimal)	0	Bronchitic change; right lower lung early	+ on admission, subsided spontaneously	Hemoptysis on admission; signs of peritoneal irritation, exploratory laparotomy revealed some free blood
13. W. W. (64718), 65. ♂	Multiple bilateral rib fractures, patient in shock	Extensive right chest wall; progression for 2 days then subsidence and recovery	+ Right (slight) early	Moderate right fluid late	Diminished aeration of right lower lung, early; persistence of lung clouding (late); hemorrhage?	0	Spontaneous recovery with expectant treatment

					rhage?		
14. I. A., (68828), 51, ♂	Fell from a chair, striking chest; multiple left rib fractures	Moderate of left posterior chest, spontaneous subsidence in 8 to 9 days	0	0	Atelectasis of left base (early), also infiltration of lung	0	
15. P. K. (62883), 58, ♂	Hit by auto; multiple right rib fractures	Moderate in axillary area of right chest; spontaneous recovery	0	0	0	0	Associated fracture of transverse process of spine
16. T. C. (64190), 58, ♂	Auto accident; bilateral multiple fracture of ribs, fracture of pubis, sacrum, and contusion of kidney	Moderate amount in left chest; early subsidence	Right early (slight), left early *considerable*	Left pleural thickening, late	Diminished aeration of right lower lobe *early;* diminished aeration of left lower lung *late*	0	Case of bilateral rib fractures and bilateral pneumothorax
17. S. G. (113975), 46, ♂	Auto accident; multiple bilateral upper rib fractures, fracture of right scapula and clavicle, head injury	Both sides of neck and right upper chest	Right + (partial) early	Fluid right, late	Atelectasis right upper lung (early)	0	
18. R. W. (117123), 25, ♂	Severe beating; multiple right rib fractures	Extensive over chest, anterior and posterior from jaw down to iliac crests; spontaneous subsidence in about a week	0	0	Minimal infiltration, right lung field (late)	0	–
19. W. G. (117493), 62, ♂	Fell off radiator and struck right chest; multiple right rib fractures	Extensive over chest, spread and involved entire body, universal, after 5 days, then respiratory distress became marked; relieved by mediastinal decompression, incision into superior mediastinum through suprasternal approach; gradual subsiddence in following week	Partial pneumothorax right base (late)	Diaphragmatic pleural thickening, bilateral (late)	–	Cyanosis dyspnea, orthopnea +	Case reported in greater detail in text
20. J. S. (118268), 57, ♂	Severe beating; multiple bilateral rib fractures	Entire chest front and back, face, lower abdomen, and back; spontaneous recovery after 4 to 5 days	0	0	Partial atelectasis or hemorrhage of right lower lung (early); pulmonary infiltration of right base (late)	0	Pneumomediastinum demonstrated on x-ray films; case reported in greater detail in text

Fifteen were blunt chest injuries. In fourteen of these there were fractured ribs, multiple in thirteen cases, and bilateral in five. One patient had no rib fracture, but had a fractured right scapula, left clavicle, and a right pneumothorax.

X-ray-demonstrable pneumothorax was present in ten cases. It was noted early in eight, one of which was bilateral, and late in two cases. In six cases it was on the same side as unilateral rib fractures; it was bilateral in a case of bilateral rib fracture; it appeared twice on the right side in cases of bilateral rib fractures, and in both of these cases the emphysema was most marked on the right side of the chest. Once it was noted without any rib fracture.

In six of the cases of unilateral pneumothorax the subcutaneous emphysema was mostly on the same side. In the other four cases the emphysema was generalized. In the nonpenetrating wounds, pleural fluid or pleural thickening was found eight times. In six instances this was a late occurrence and was absent on the early x-ray views. It was on the same side as the unilateral rib fractures three times, was bilateral in the case of universal emphysema, appeared in the case without rib fracture, and was seen three times in the cases of bilateral rib fracture.

Pulmonary infiltration suggestive of pulmonary interstitial hemorrhage, diminished aeration, or patchy atelectasis was seen in nine cases. It was seen late in four lungs (early x-ray pictures failed to show it). Early lung changes occurred in eight lungs. Partial collapse of the lung occurred twice. Respiratory embarrassment was present in five cases. It was marked in one fatal case and developed in the second fatal case, possibly playing a part in the mortality. In the other three cases it was marked only in the case of universal emphysema.

The emphysema was extensive in twelve cases, moderate in six, and slight in two. Active treatment toward relieving the emphysema was necessary once and might have been of value in two of the fatal cases (death was probably due to the associated injuries).

DISCUSSION

Numerous instances of interstitial pulmonary, mediastinal, and subcutaneous emphysema following a variety of causes have been reported in the literature. For the most part, with the exception of cases of direct chest trauma, these cases have been explained or proved to be caused in the manner described under Mechanism 2.

Cowart[5] described two cases of mediastinal emphysema following chest contusions with spontaneous recovery in each. Lilienthal[6] quoted a case from Dr. Fred Zimmer, where generalized subcutaneous emphysema after multiple rib fractures with marked dyspnea was successfully treated by open thoracotomy. Friedman and King[7] reported a case of mediastinal and extensive subcutaneous emphysema following an automobile accident. Dyspnea was marked at first. Oxygen was given and spontaneous recovery ensued.

Cases have occurred after general anesthesias. Thornton, Jr., Adams, and Livingstone[8] reported a fatal case. At autopsy, marked mediastinal and pulmonary interstitial emphysema were demonstrated. Another fatal case was cited by Barret and Thomas.[9] A convulsion complicated an endotracheal gen-

eral anesthetic. General subcutaneous emphysema rapidly developed, and death ensued before effective therapeutic measures could be instituted. These authors also reported another case of emphysema successfully treated by needle aspiration and general measures.

Bronchoscopy has, on a number of occasions, been followed by a rapidly developing generalized subcutaneous emphysema, dyspnea, and cyanosis. Jones[10] reported two patients, desperately ill, but each recovering after needling and general supportive measures. Hammond[11] incised the neck, opening the perivascular spaces and the superior mediastinum with a successful outcome in a severe case. Fisher and Macklin[12] reported a fatal case following a peanut inhalation, with resultant atelectasis and subsequent bronchoscopies. At autopsy, mediastinal and interstitial pulmonary emphysema with rupture of multiple alveoli were demonstrated.

Cases complicating influenza pneumonia,[13] bronchopneumonia,[14] and asthma[15] have been collected and published.

The literature is a vast one, and only a few illustrative reports are quoted. However, the underlying principles of disturbed physiology appear to be similar in the numerous instances of this clinical syndrome.

TREATMENT

The method of approach in curing subcutaneous emphysema will depend upon the type of injury and its extent.

1. In true mediastinal emphysema, mediastinal decompression should be employed. This can best be done by incision over the jugulum and adequate drainage of the air from the mediastinum. Occasionally needle aspiration, suprasternally or parasternally, may suffice.

2. A needle or trocar may be introduced into the pleural space, and underwater drainage established. This will interrupt the further extension of air across the pleural space into the subcutaneous tissues.

3. Pneumothorax with moderate positive pressure may be used to compress the lung, and thus prevent further air leakage from it.

4. Thoracotomy with suture of the parietal pleura alone may be enough.

5. Suture of lung laceration may be done. This will be necessary rarely.

6. Combination of these methods, in cases of extensive and rapidly forming emphysema, may be utilized.

7. Expectant treatment may be enough when careful observation shows there is little or no progression.

The course of events may be relatively slow, and adequate treatment may be decided upon after ample deliberation. On the other hand, progress may be very rapid, and one's resources may be severely taxed to apply an effective combination of methods. Adequate oxygen concentration and a free airway must be assured.

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CARCINOSARCOMA OF THE MAMMARY GLAND

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IN A comprehensive review, Saphir and Vass[1] assembled the available reported cases of carcinosarcomas, and tabulated 153 according to the sites of occurrence. They concluded that perhaps only three or four of them might be designated as true carcinosarcomas. Among the thirty-two cases recorded as primary in the mammary gland, Saphir and Vass apparently believed none to be true carcinosarcomas; they considered twenty-one as "duct carcinomas." Since then Harrington and Miller,[2] Smithy,[3] Govan,[4] Mosto,[5] and Adair and Herrmann,[6] have reported cases of carcinosarcoma of the mammary gland. In this paper an additional case is presented, and because of the rarity of the neoplasm and the theoretical implications, the data are recorded in some detail.

REPORT OF CASE

A 43-year-old Negro woman was admitted to the University of Oklahoma Hospitals, April 8, 1944, complaining of a mass in the left mammary gland, first noticed four months previously, and twenty pounds loss in weight during the previous year. The patient stated that two months before admission, she slipped and fell on ice, striking the left breast. Following the fall the mass became painful and increased in size. On March 18, 1944, a physician incised the mass and "some pus and a large amount of blood" were obtained. The mass remained tender and the wound failed to heal, draining a brown purulent material.

At the time of admission the patient was obese and did not appear ill. The temperature was 100.6° F. In the upper inner quadrant of the left mammary gland there was a firm, warm, tender mass 8 cm. in diameter attached to the overlying skin. From an elevated, ulcerated area of the skin 2 by 2 cm. over the mass, a foul sanguinopurulent liquid exuded. The lymph nodes in the axilla were not palpable. The remainder of the physical examination yielded no pertinent information.

Urinalysis gave essentially negative results. The red blood cell count was 4,580,000; the hemoglobin content was 15.2 Gm.; the white blood cell count was 8,100, with polymorphonuclears 55, lymphocytes 40, monocytes 4, and eosinophiles 1 per cent. The Mazzini test of the blood was negative. A roentgenogram of the chest disclosed no evidence of pulmonary metastasis. A biopsy from the ulcerated area revealed a rapidly growing malignant neoplasm.

On April 14, 1944, a radical mastectomy was performed. The postoperative course was uneventful, except for some separation of the incision which necessitated a skin graft on May 8. The patient was discharged on May 19. Subsequently, she was given roentgen radiation over a period of twenty-seven days (May 31 to June 26) to four fields, two over the left upper thorax, one over the left axilla, and one over the left supraclavicular region (total dose 1,600 r. to the two anterior fields and 2,200 r. to the two fields over the axilla and supraclavicular regions; distance 50 cm.; 170 kv. over the two anterior fields and 200 kv. over the left axilla and supraclavicular regions; 18 ma.; Thoraeus filter).

When seen on Jan. 3, 1945, the patient had gained fifteen pounds in weight. There was some edema of the left arm and no evidence of any local recurrence. A roentgenogram of the chest disclosed numerous round areas of increased density scattered throughout both lung fields interpreted as pulmonary metastases. The patient died in another hospital on March 11, 1945. No necropsy was performed.

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The biopsy specimen consisted of two mottled brown and yellow-gray fragments of tissue together 2.5 by 1.5 by 0.8 centimeters. Microscopic preparations stained with hematoxylin and eosin disclosed sheets of large neoplastic cells with marked variation in size and shape of the nuclei and many in a state of division. In places, clusters of apparently epithelial cells were seen surrounded by cells in a streamlike arrangement within a fibrillar ground substance into which faded the cytoplasm of the cells. Occasional cells had giant proportions with several nuclei. Areas of necrosis and hemorrhage were frequent, and a broad zone of fibrinopurulent exudate covered part of one surface.

The specimen obtained at operation consisted of the left mammary gland with the underlying pectoral muscles and axillary contents. The nipple was inverted, freely movable, and centrally located in an elliptical portion of dark brown skin 20 by 15 centimeters. In the upper inner quadrant in the skin overlying a fluctuant mass 8 cm. in diameter, there was an ulcerated area 2 by 2 cm. with everted, heaped-up margins. On the cut surfaces this led into a craterlike cavity 6 cm. in diameter filled with green, yellow and brown tissue debris surrounded by a soft, pale yellow-gray margin of neoplastic tissue 1 cm. wide (Fig. 1). Continuous with the necrotic area, there was a gray-pink neoplastic field 4 by 3 centimeters. Detectable lymph nodes in the axillary content were in great part replaced by adipose tissue, and there seemed to be no neoplastic involvement.

Fig. 1.—Appearance of the growth on the cut surfaces. An ulcerated area in the everted, heaped-up margins leads to a craterlike cavity 6 cm. in diameter filled with tissue debris and lined by neoplastic tissue.

In microscopic preparations stained with hematoxylin and eosin, representing many parts of the growth, there were sheets, columns, or nests of neoplastic epithelial cells in a stroma composed of neoplastic cells with large vesicular, round, oval, or irregularly shaped nuclei in a streamlike or interlacing arrangement. The cell nuclei varied markedly in size, and a number were seen in a state of division. The cytoplasm of these cells faded into a fibrillar ground substance similarly arranged in streams or whorls. The epithelial elements in the obviously nonepithelial stroma were composed of cells with large, vesicular, deeply stained nuclei with pink or lavender stained cytoplasm having clearly discernible borders or within a halo of cytoplasm. Some of the cells had huge nuclei or multiple nuclei with some in multicentric cell division. Although the epithelial and nonepithelial elements blended imperceptibly in places (Fig. 2), elsewhere the stroma was obviously provided by the nonepithelial growth (Fig. 3). Areas of necrosis and hemorrhage were frequent and extensive. The

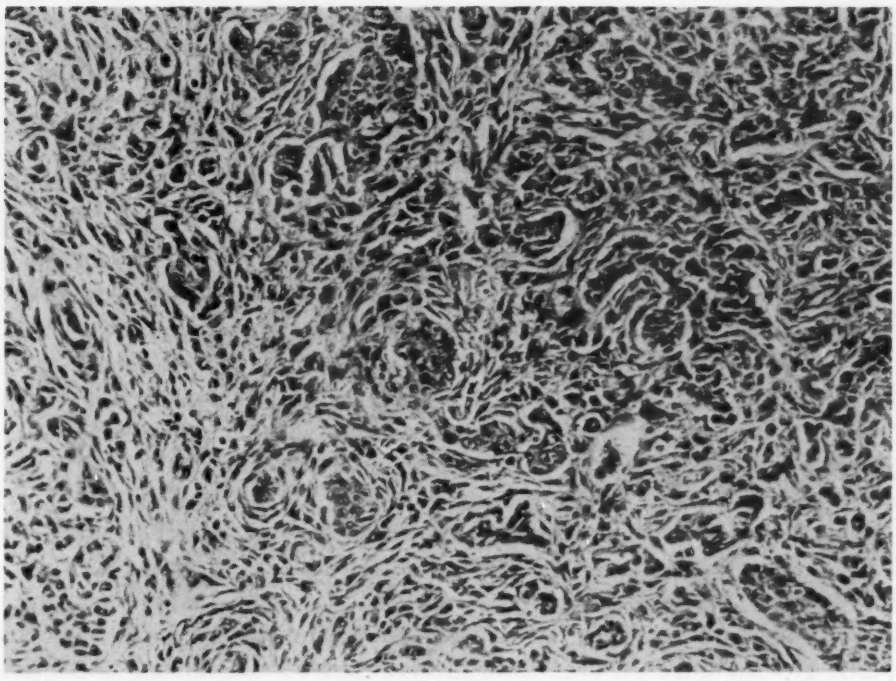

Fig. 2.—The cancerous growth is composed of epithelial elements, carcinoma, in a sarcomatous stroma. The two tissue elements in places blend imperceptibly (×150).

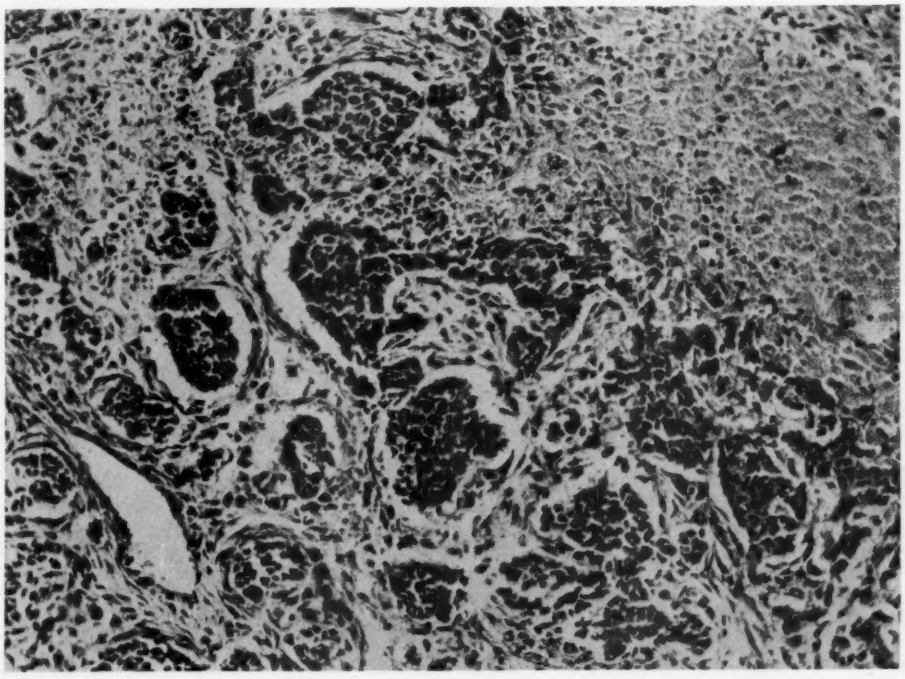

Fig. 3.—The epithelial elements of the cancerous growth are clearly discernible from the sarcomatous stroma (×150).

distribution of the obviously epithelial and nonepithelial elements varied in different portions of the growth. In the periphery of the growth in places there appeared to be a condensation of the fibrous connective tissue. Occasional groups of intact acini and ducts of the mammary gland were seen surrounded by a loose connective tissue fading into the neoplastic tissue. Preparations from the axillary lymph nodes disclosed marked adipose tissue replacement of the lymphatic pattern and no neoplastic involvement.

COMMENT

There is ample evidence that cancerous growths composed of both carcinomatous and sarcomatous elements occur. In the mammary gland, they have been seen in mice,[7] rats,[8] and men. Neoplastic proliferation of the epithelium in the mammary gland seems to furnish an incentive for neoplastic growth of the stroma as in fibroadenomas. A pure fibroma of the mammary gland is exceedingly rare.[9] Therefore, the interpretation as to the origin of carcinosarcomas needs not necessarily be complicated, although the possibilities enumerated by Harrington and Miller[2] must be kept in mind. Perhaps the simplest concept is to assume that the epithelial element, the carcinoma, is dominant and that the connective tissue element, the sarcoma, is subservient and forms the stroma. Carcinoma always stimulates the surrounding connective tissue to provide a stroma for it. In this instance the stimulation was such that a sarcomatous stroma was produced. The sarcoma, on the other hand, provides its own stroma, and, therefore, is unlikely to initiate the growth of a carcinoma. According to this concept, the carcinomatous metastases might induce the proliferation of a sarcomatous stroma while sarcomatous metastases would be free of carcinoma. Unfortunately, in our case there was no opportunity to study the structure of the metastatic growths.

SUMMARY

A carcinosarcoma of the mammary gland is reported in a 43-year-old Negro woman. The morphologic appearance of the rare cancerous growth suggested that the epithelial element, the carcinoma, was dominant and that the connective tissue element, the sarcoma, formed the stroma.

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A NEW MATERIAL AND TUBE DESIGN IN GASTROINTESTINAL INTUBATION

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PROLONGED use of the rubber stomach or intestinal tube passed intranasally is frequently the cause of bitter complaint on the part of the patient due to obvious irritation in the nasopharynx and esophagus. The natural quality of rubber material is a high degree of stickiness, a property which makes it useful in tires to prevent slipping, but truly uncomfortable and irritating in the nose and throat.

Holinger and Loeb[1] recently reported four cases of severe infection of the cricoid cartilage following ulceration of the postcricoid mucosa in the anterior wall of the esophagus caused by the irritation of intranasal tubes made of rubber. A survey of the literature reveals twenty-four cases of serious pharyngeal infections following the usual rubber intubation procedure through the nose.

Repeated trial and observation have shown that intestinal intubation at times is a lifesaving procedure. Eliason and Welty,[2] in a ten-year survey of intestinal obstruction, concluded that where suction drainage by means of the Jutte, Levine, or Miller-Abbott tube was carried out in 124 of 292 cases (as an adjunct in treatment of obstruction from all causes) it was of value in 80 to 90 per cent of cases. In fact, in 25 per cent of the cases in which suction drainage was carried out, no subsequent operative procedure was required. They felt that it was particularly valuable in obstructive cases developing postoperatively where fresh adhesions are the basis for the obstruction. In most of these cases intubation eliminates the necessity for a subsequent operation. The statistics they presented are convincing proof of the lifesaving possibilities of intraintestinal intubation. When the Miller-Abbott tube was used for intraintestinal suction in 1937 for the first time, the 31 per cent mortality that existed with the use of the intragastric tube from 1934 to 1937 was reduced by 50 per cent. In sixty-five cases where intraintestinal intubation was used after 1937 the mortality was only 15 per cent. Intubation can prevent surgical intervention in a partial intestinal obstruction apparently by reducing intraintestinal tension.

It is because such important uses have been firmly established for intestinal intubation that a new material was sought that would justify more frequent and early use of the intranasal tube. This is important since there is a great deal of reticence among doctors to use an intranasal tube except in serious circumstances. This is understandable since there is a marked discomfort inflicted on the patient by the use of the rubber tube. The present report deals

Tubing provided by Don Baxter Company, Glendale, Calif., and Clinical Plastic Products, Los Angeles, Calif.

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with a tube made of plastic material which reduces to a negligible degree the nasal and pharyngeal discomfort. This material has so little irritative effect that its early use in gastric or intestinal difficulties due to irritation or stasis is urgently recommended since it will cause little or no irritable complaint from the patient.

Of all plastic materials tested, the one that proved to be most useful and least irritating was a smooth transparent plastic of the Koroseal type. From this material, modifications of the stomach and intestinal tubes now made of rubber were developed and are discussed herein.

All plastic tubes to be described are of a smaller caliber than rubber tubes (14 to 16 Fr. diameter) and can readily be passed through the nose. The tubes are of a clear colorless transparency, have a moderate amount of pliability with a satisfactory amount of elasticity, and above all possess a smooth glasslike slickness.

ADVANTAGES OF PLASTIC OVER RUBBER

The advantages of this plastic material over rubber are fivefold:

1. Plastic tubes have a smoother surface than rubber, thereby reducing friction in the nose and throat while passing the tube, as well as minimizing the irritation on the pharynx while swallowing after the tube is in place.

2. The smoother plastic material cannot kink or twist on itself tightly enough to obstruct the flow in either direction. As many as three tight knots were experimentally tied in a length of tubing, yet suction could be maintained to carry on the withdrawal of intestinal contents. This is due to the negligible amount of stickiness of plastic material whereas rubber has a marked amount.

3. The colorless transparency gives continuous visualization of the stomach or intestinal contents as siphoned out. The nature of the material can in this way be continuously observed and alteration in treatment made frequently. With opaque rubber tubes, definite periods of collection of intestinal contents in bottles are necessary to see the type of drainage taking place.

4. The absence of deterioration of plastic when in contact with oil or ointments permits the use of ointments having greater lubricating properties in passing the tube. More tenacious ointments can also be applied after the tube is in place. This adds considerably to the comfort of the patient since it minimizes the irritation of the tube on the nasopharyngeal tissues. The only types of lubricant that can be used on rubber tubes are water-soluble types and these tend to disappear rapidly.

5. The inexpensive cost permits expendability. The cost of this tubing is much less than for an equal length of rubber tubing. In time it will be economically feasible to discard the plastic tube after one use. The discard of gastric and intestinal tubes will save much time and labor which is at present expended needlessly in the cleaning of the tubes by supply room personnel after each use. The use of a fresh noncontaminated tube on each new patient will also mark a step forward in medicine. At times re-used rubber tubes have such disagreeable odors that it is not very convincing to the patient when he is told that the ill-smelling tube about to be used on him has been sufficiently

cleansed in order to be used safely again. There is no odor to the plastic tube such as rubber tubes possess.

GASTROINTESTINAL TUBE

This design of a plastic gastrointestinal tube differs somewhat from the present types made of rubber. It is a single-lumen plastic tube ten feet long with a weighted balloon attached to its end. The balloon is weighted by being partly filled with 5 c.c. of liquid metallic mercury. The balloon is a separate chamber and has no connection to the lumen of the tube, nor does it require any inflation or deflation after passage through the nose. The end of the tube is weighted in this manner so that gravity and peristalsis will move the tube through the intestinal tract in the same manner that a bolus of food is swept along. X-ray studies clearly show the location of the mercury-filled balloon at all times.

Proximal to the attachment of the balloon are perforations directly into the lumen of the tube into which the intestinal contents are sucked.

The distal tip of the tube is made of a loop of small caliber plastic tubing which is soft and flexible. This tip is unlike a metal ball and has no traumatic effect either in passing through the nasopharynx or on the gastrointestinal mucosa. Perforations in the tip permit suction of gastric or intestinal contents from the distal end of the tube as well as from the area behind the balloon.

The simplicity of this tube eliminates a good deal of nurse and doctor care, since once it is passed there is no necessity of constantly watching the tube for the proper time to inflate the balloon in order to move it through the intestinal tract. To pass the tube the patient is placed in a semisitting position with the head tilted back. The balloon and tubular tip is then passed through the nasal orifice. The mercury in the balloon runs into the dependent part of the chamber when it reaches the back nasopharynx and aids in directing the tube down the esophagus. After the passage of the tube into the stomach, or to the 1½ foot mark, the patient is placed on his right side. In a short period of time, gravity or peristalsis pulls the weighted balloon into the pylorus (see Fig. 1). Having passed through the pyloric sphincter it readily moves into the duodenum. In repeated trials the tube has been inserted up to ten feet into the intestinal tract in a much shorter period of time than the presently used rubber tubes require.

In over 110 cases of intestinal intubation with this plastic tube it has been shown that there are many advantages in simplicity of handling and there is a remarkable absence of discomfort to the patient after as long as fifteen days of constant use.

STOMACH IRRIGATION TUBE

This design of a stomach irrigation tube consists of a double-lumen tube in which a narrow tube is inserted through and beyond a larger diameter tube. Both tubes are of plastic material. The smaller tube extending beyond the large tube has perforations only at its distal end so that cleansing fluids instilled through it will run directly into the stomach. The larger tube has per-

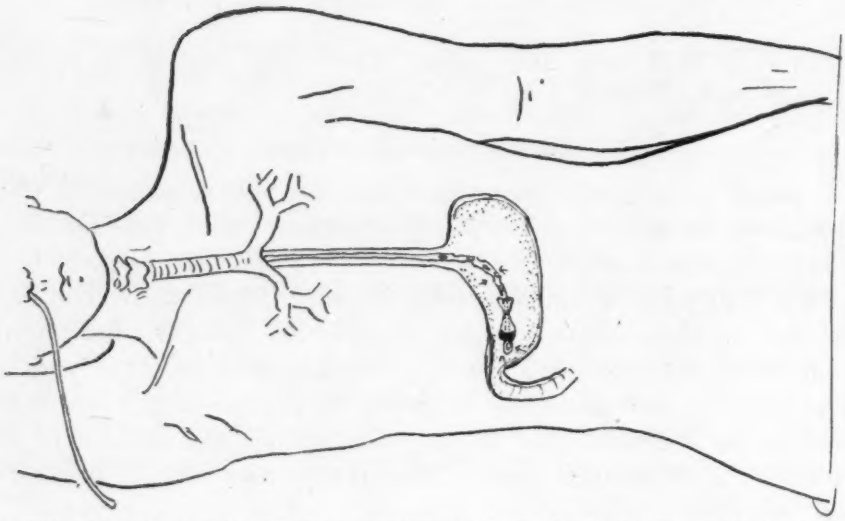

Fig. 1.—Weighted tip of tube showing structure; gravity tends to direct tip toward pylorus.

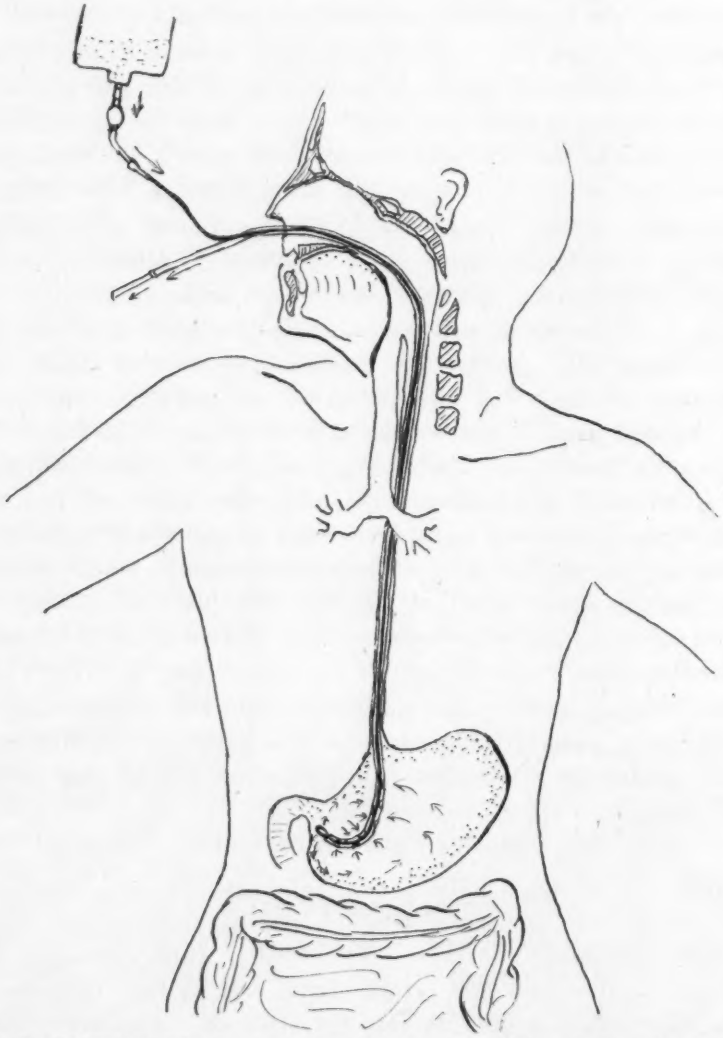

Fig. 2.—Irrigation tube in stomach indicating circulation of cleansing fluid.

forations proximal to its end so that suction applied to it sucks back the washings of the cleansing fluids ejected from the smaller tube (see Fig. 2).

This tube is especially useful in those cases requiring repeated *lavage* over a period of time, where it is necessary to spare the patient as much discomfort as possible due to debility or shock. It has been used with good cleansing results in postoperative nausea and vomiting, partial intestinal obstruction, barbiturate poisoning, gall bladder distress with nausea and vomiting, and as a cleansing procedure following food poisoning. Recently it has been used with success as a continuous irrigation of the stomach for four days in a case of severe nephritis in which anuria was imminent. The patient survived the acute phase of the disease.

By this tube arrangement cleansing fluids, such as bicarbonate of soda or saline solution, are continuously instilled into the stomach while a Wangensteen suction drains the washings back. This is a true irrigation device. Furthermore, it can be set up and left functioning without constant nursing attention. Irrigation with the present single-lumen tube requires the pushing back and forth of the column of material already in the tube with each use of the syringe.

One liter or more of cleansing solution is attached to the small lumen tube and allowed to run in by gravity, while an alternate suction or Wangensteen suction is attached to the larger suction tube. Irrigation and lavage in this manner is very gentle and is carried out without the discomfort to an already irritable stomach of spasmodic gushes of fluid into it. It also offers the advantage of continuous operation day and night, without disturbing the patient, simply by adding fluids to the irrigation bottle. Due to its negligible discomfort to the patient, the device described here has uses other than that of an irrigation apparatus where prolonged use of a nasal tube is required. It has been used in gall bladder drainage by allowing a solution of magnesium sulfate to drip through the small tube into the duodenum, while the bile is drained back through the larger tube.

It has been used for intragastric as well as intraintestinal feeding for periods lasting up to ten days. Feeding can be more easily accomplished by gravity feeding through one lumen while medication is administered through the other tube. This eliminates the need to disconnect the tube to give medication.

SUMMARY

Stomach and intestinal tubes made of plastic material have been extensively used and found to be comparatively free of discomfort compared to rubber tubes.

A simplified tube used for intestinal intubation is presented to indicate its greater ease in handling.

A two-way stomach tube that permits continuous irrigation and *lavage* is discussed. Other uses for such a tube are mentioned.

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ELECTRIC LIFT

An Aid in the Treatment of Decubitus Ulcers

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REPORTS of surgical treatment by excision and plastic closure of decubitus ulcers in paraplegic patients indicate an increasing number of successful results. Most of the reports to date come from military hospitals where these patients were first received and treated. Eventually all veterans with spinal cord injuries will of necessity be admitted for treatment to the several paraplegic centers in the Veterans Administration Hospitals. It is in such centers that surgical treatment of bed sores poses a formidable problem as far as the local treatment is concerned.

Suitable and frequent changes of dressings are a necessity and are extremely difficult to accomplish because the patients are paralyzed and the lesions are most frequently found about the pelvic girdle. Extensive bed sores can be compared with deep burned lesions and require properly applied pressure dressings. Uninvolved bony prominences must be given special attention while a dressing is applied, and should be well padded to prevent additional ulcerations. After plastic closure has been accomplished, voluminous pressure dressings must also serve as a splinting device to produce at least partial immobilization. At least two assistants are needed during application of a dressing and even then, the process is tedious and the dressing not entirely satisfactory.

A well-organized and properly equipped dressing room is indispensable on a large service where many pre- and postoperative dressings are done each day. The main feature of this dressing room is the electric hoist which enables one doctor or one nurse to change an extensive dressing properly without exertion. The results where these hoists have been used were so uniformly gratifying and the acceptance by the patients of this method so enthusiastic, that it was thought advisable to present it graphically in detail. The accompanying illustration is self-explanatory.

The procedure usually carried out is as follows: The litter with the patient is wheeled into the dressing room to a position directly under the hoist. With the patient in prone position, a 9 inch belt is placed under the chest, a 6 inch belt under each thigh, the snaps secured, and the separator placed between the two cables that support the thighs. The lever is pulled that raises the patient to the desired height and the old dressings are removed. Aseptic technique is easy to carry out while the patient is suspended. When the patient is redressed, he is lowered to the litter by pulling the proper lever of the hoist.

Among the variations from this usual procedure, the following may be noted: At times, a patient in bed or on a Stryker frame may be brought in directly without placing him on a litter. It is obvious that the door to the dressing room must be wide enough to admit a hospital bed. Although the prone

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position is most satisfactory, should a spastic condition of the patient require that he be raised with the hips flexed, he may be placed into the apparatus lying on his side, a pillow between the knees, one belt around both thighs, and the upper belt around the chest. If spasms are too frequent to allow the patient to remain suspended during the entire change of dressing, he is raised long

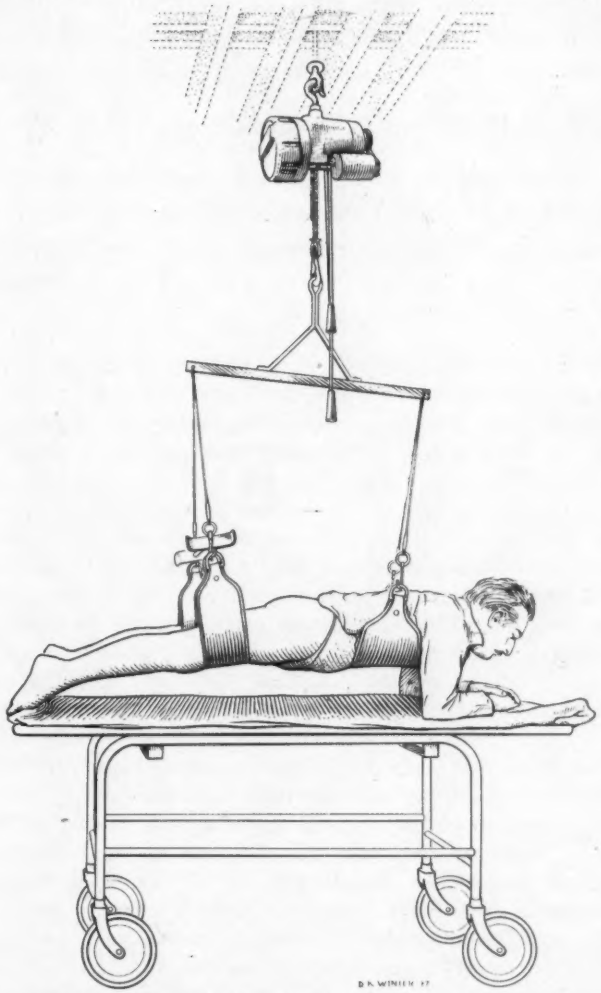

Fig. 1.—The paraplegic patient is elevated to a sufficient height from the cart allowing direct access to all parts of the pelvis. Local treatment of the decubitus ulcers and proper application of dressings are markedly facilitated. The electric lift shown is ⅛ ton "Budgit" hoist manufactured by Manning, Maxwell & Moore, Inc., Muskegon, Mich.

enough to remove the outer bandage material and lowered for the necessary observation and treatment and raised again to permit reapplication of the dressing.

SUMMARY AND CONCLUSIONS

1. The importance of proper dressings should be recognized as a very important part of the surgical treatment of decubitus ulcers. Because of the paralytic condition present and the location of the ulcers, proper application of dressings is extremely difficult to attain.

2. A dressing room featuring the use of an electric hoist is presented which markedly reduces the difficulties of handling the patient and allows easy application of dressings.

3. With slight variations, this type of hoist may also be used in the operating room for immediate postoperative dressings. It may be anticipated that this method may well serve other uses, such as the treatment of burns about the pelvic girdle and in certain orthopedic conditions requiring unobstructed access to the pelvic region.

Case Reports

ASSOCIATED PANCREATIC NECROSIS AND BILIARY DISEASE

A Case Report

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THE case we are presenting for review is of interest in that: first, the problem of acute pancreatitis associated with cholelithiasis is presented; second, the nonsurgical removal of persistent postoperative stones in the common duct is accomplished.

CASE REPORT

M. B. (Clinic No. 124482), a 38-year-old Jewish housewife, was first referred to the Mason Clinic, April 9, 1946, with the chief complaint of intermittent, severe, right upper quadrant pain associated with nausea and vomiting of four days' duration.

The family history was noncontributory. She had suffered no previous injuries or serious illnesses in the past; she had had no previous surgical operations. She had been perfectly well until five years prior to admission. At that time she first noticed loose stools, especially in the morning. The stool was not watery, but was soft and bulky. The increase in bowel movements gradually continued until two years prior to admission she was having seven to ten soft loose stools daily, and at the same time noticed onset of right upper quadrant pain with nausea and vomiting which spontaneously subsided after a few bouts. There was an increase in the frequency and severity of the attacks until for the four days prior to hospital admission she had suffered continuous attacks.

Systemic review revealed no significant facts except that the patient had always been obese, for many years having weighed over 200 pounds.

Physical examination revealed a moderately ill, obese woman, 5 feet 5 inches tall and weighing 211 pounds. Temperature on admission was 98° F., pulse 70, respirations 16, and blood pressure 150/90. There was an icteric tinge to the sclera, and a very slight icterus of the skin. Chest examination was negative. The abdomen was obese. There was tenderness in the right upper quadrant and in the epigastrium, but because of the thickness of the abdominal wall, neither muscular rigidity nor masses could be demonstrated. The remainder of the general physical examination revealed no significant findings. The significant laboratory tests were an icterus of 55, bile in the urine, demonstrated multiple stones in the gall bladder, and a normal leucocyte count.

Three days after completion of the laboratory results, and a total of six days after the first visit, she was admitted to the hospital in an emergency with severe exacerbation of the right upper quadrant pain. The last attack had been of nine hours' duration, associated with continuous nausea and the vomiting of greenish bile-stained material. The attack was the same as those previously described, except with increasing severity and radiation to the back. Stools during this period had been persistently white with dark urine. A repeated complete physical examination showed no change from the previous examination except that the temperature on admission was 95.4° F. (checked), pulse 60, respiration 16. There was more marked tenderness in the right upper quadrant and epigastrium and some tenderness in the left upper quadrant. Repeated blood and urine examination revealed no

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essential change from the previous reports except an increase in the leucocyte count to 13,000 with 84 per cent neutrophiles; the icterus index had increased to 75 units.

The clinical impression was that the patient suffered from (1) an acute exacerbation of chronic cholecystitis with demonstrated stones, and (2) common bile duct obstruction due to lithiasis, and with extreme obesity to complicate the surgical management of the condition. On admission she was given glucose infusions plus demerol hypodermically for pain. The following day the temperature ranged between 97 and 98.6° F., the pulse from 70 to 80, and the patient showed clinical improvement.

Choledochostomy and cholecystectomy were advised. On April 18, 1946, nine days after the first examination, the patient was operated upon under inhalation anesthesia. When the peritoneum was opened there were immediately apparent multiple miliary areas of fat necrosis (Fig. 1), averaging 5 to 10 mm. in diameter, and one of these areas was removed for biopsy and photograph. These covered the greater omentum, the gastrocolic omentum, and the fat around the duodenum and the duodenohepatic ligament. The head of the pancreas was the size of a baseball, was diffusely firm, and was intimately adherent by fresh adhesions to the overlying colon and other structures. The gall bladder was moderately distended with a slightly thickened wall, but without exudate. It was packed with multiple,

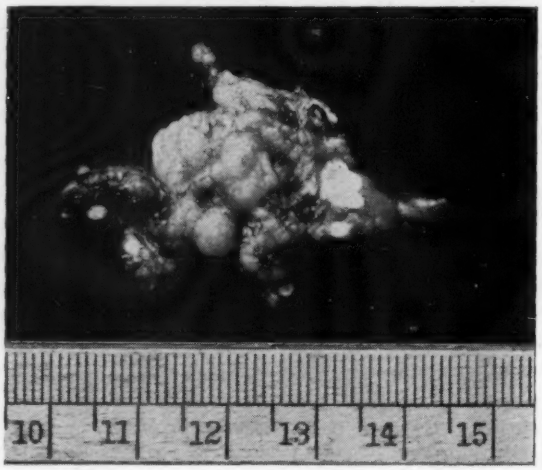

Fig. 1.—Biopsy of omentum showing fat necrosis. Microscopic study was reported as caseous necrosis of omentum.

small faceted stones with several of these impacted in the cystic duct, which was long and small. The common duct was not visualized nor was it openly explored, because of the edema of the duodenohepatic ligament, the encroachment upon it by the enlarged pancreas, and the critical state of the patient. During the induction stage of the anesthetic the patient vomited bile and gastric secretion; she had a dusky color from this point throughout the remainder of the operation. It was felt that she may have aspirated some of the gastric content. This, plus the proved high mortality rate in acute pancreatic necrosis and the individual technical problem here involved, influenced us to resort to simple cholecystostomy. By removing the stones from the cystic duct, indirect drainage of the common bile duct was provided. To insure that this was accomplished a continuous flow of common duct bile into the gall bladder via the cystic duct was demonstrated. The gall bladder was then drained with a No. 12 Pezzer catheter and brought out through a stab wound in the right flank. The patient's condition upon completion of the operation was satisfactory, with a stable blood pressure and pulse, and with a fair amplitude of respiration.

A serum amylase determination, neglected preoperatively, was made immediately after the operation, and was found elevated to 432 units. Although considered as critically ill postoperatively, the patient made an uneventful convalescence. During this period the

Pezzer catheter continued to drain from 100 to 300 c.c. of moderately clear bile daily, although cramps ensued if the tube was clamped. There was a rapid diminution in the icterus so that by the ninth postoperative day the icterus index was reported as 19 and the serum amylase as 122. The stool examination showed an excess of neutral fat present. Microscopic report of the biopsy of omentum was "caseous necrosis of the omentum."

The cholangiogram at this time (Fig. 2) showed multiple nonopaque areas in the distal third of the common duct which had the appearance of stones; the oil entered the duodenum readily and the maximum diameter of common duct was 13 mm. Observation films at five and ten minutes and one hour showed residual fluid in the common duct. This was due in part to the fact that some emptied from the gall bladder. It was noted that injection of the oil caused severe, upper right quadrant pain.

Fig. 2.—Cholangiogram on twelfth postoperative day showing multiple nonopaque areas in distal third of common duct.

The patient was discharged from the hospital on the fifteenth postoperative day with a final diagnosis of chronic cholecystitis and cholelithiasis, residual choledocholithiasis, and acute pancreatic necrosis. The Pezzer catheter was connected to an infant hot-water bottle to collect conveniently the biliary drainage, and the patient was instructed to irrigate the tube daily with normal saline solution to insure against encrustation. At home the patient continued to regain strength and was much improved except that if the catheter was clamped off or became plugged at any time, she immediately suffered exacerbation of the right upper quadrant pain which was relieved by opening the tube. A second stage choledochostomy to remove the residual stones was planned after sufficient time should have elapsed for the pancreatitis to subside.

It was at this time that Harris and Moncus[1] suggested the injection of 1:500 solution of nupercaine in normal saline solution for relief of spasm of the sphincter of Oddi, and reported one instance in which this method had allowed passage of a residual common duct stone.

On May 15, twenty-nine days after operation, the catheter in the gall bladder was injected with 30 c.c. of 1:500 nupercaine solution in normal saline solution, and irrigated

with normal saline solution afterward without pain. Thirteen days later the patient reported to the hospital at 9 P.M. with a severe episode of right upper quadrant pain similar to that she had had preoperatively. Nupercaine solution, 30 c.c. injected into the Pezzer catheter promptly relieved the pain and a third injection of nupercaine solution carried out thirteen days later produced no pain.

At this time a repeat cholangiogram (Fig. 3) was obtained, and showed the maximum diameter of the common duct to be 12 mm., with a small nonopaque area in the distal common duct. In fifteen minutes, 20 per cent of the oil had escaped into the duodenum, and in thirty minutes, 50 per cent. The multiple, nonopaque shadows previously noted had disappeared except for one remaining. Irrigation with nupercaine was carried out at ten-day intervals and thirty days after the second cholangiogram a third x-ray series demonstrated the diameter of the common duct to be reduced to 10 mm., and the previously noted

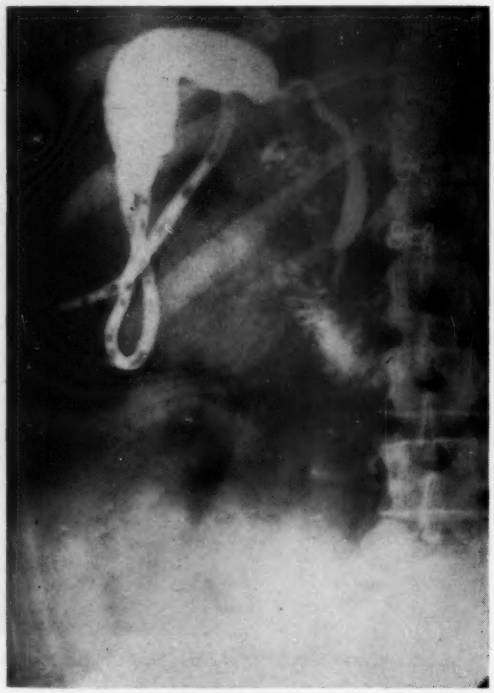

Fig. 3.—Cholangiogram taken after three irrigations of cholecystostomy tube with 1:500 nupercaine in saline solution carried out over a twenty-six day period, showing one remaining nonopaque area in distal common duct.

Fig. 4.—Final cholangiogram taken thirty days after that shown in Fig. 3, during which period the Pezzer catheter had been irrigated with nupercaine solution three more times. The filling defects shown previously are no longer present and the diameter of the common duct is reduced to 10 mm.

filling defect no longer present. Oil entered the duodenum immediately, with 60 per cent emptying in ten minutes. The tube was clamped off during the day and biliary drainage at home which had been averaging 300 c.c. daily, promptly diminished to only 10 or 15 c.c. during the night. The patient was without distress. The Pezzer catheter was removed on Aug. 1, 1946, three and one-half months postoperatively. The patient continued well for the ten months following that time.

Comment.—The association of inflammatory disease of the pancreas and infection of the biliary tract is recognized, but the teleological relationship is not satisfactorily understood. Investigators have reported biliary tract disease in from 16[2] to 81 per cent[3] of cases of pancreatitis, with an average incidence in

large series of 55 per cent.[4, 5] The exact evaluation of the relative involvement of the biliary tract and of the pancreas has assumed greater importance in the last ten years since it has been demonstrated that under conservative treatment the mortality of acute pancreatitis is less than with emergency surgical intervention. The development of a reliable and rapid determination of the serum amylase has afforded us a valuable guide to severity in early cases. On our service we feel that acute cholecystitis should be handled as a surgical emergency and operation deferred only for necessary preparation or for nonsurgical complications. Since associated acute pancreatitis is one of the latter, we have the additional rule that a serum amylase determination be made before emergency laparotomy for the acute crisis of the upper abdomen. Like many rules that are made to be broken, we slipped in this instance—a mistake that fortunately turned out satisfactorily.

The second point of interest concerns the stones necessarily left in the common duct at the time of emergency cholecystostomy. Previous attempts to remove overlooked or secondary stones in the duct, usually following primary choledochostomy, by chemical means have in most instances been fraught with failure until Harris and Moncus,[1] applying a new principle, suggested relaxation of the sphincter of Oddi, rather than chemical dissolution of the stones. The practical application of this principle is successfully demonstrated in the case herein presented. Whether success following pressure irrigation with nupercaine solution should be attributed to the relaxation of the sphincter by the anesthetic agent or whether the anesthetizing of the ductal tree permits greater pressure to be used remains unproved. Preliminary studies on a small series indicate a lower perfusion pressure following nupercaine injection, but no lessening of the pain threshold, thus indicating that relaxation of the sphincter of Oddi is the mode of action. Further studies on this are continuing.

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Recent Advances in Surgery

Conducted by Alfred Blalock, M.D.

MESENTERIC CYSTS

A Review of the Literature and Report of a Calcified Cyst of the Mesentery

Arkell M. Vaughn, M.D., William M. Lees, M.D., and James W. Henry, M.D., Chicago, Ill.

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CYSTS of the mesentery have been described as the rarest of all abdominal tumors,[20] notwithstanding a score or more yearly case reports in recent years. The preoperative diagnosis of this condition is a difficult problem and requires ingenuity and experience. Because of the rarity of correctly diagnosed cases preoperatively, we should like to present this case and bring the literature up to date in so far as it is possible, with poor scientific communications between countries isolated by World War II.

HISTORY AND INCIDENCE

The first observation of this entity was made by the Florentine anatomist, Benevieni in 1507 who called these cysts found at autopsy, "anatomic curiosities."[10] In 1842, Rokitansky[38] gave the first recorded description of a chylous cyst. Tables I and II record chronologically contributions of various authors on the subject and their estimates as to the incidence of mesenteric cysts. Further incidence is illustrated by the following:

1. Judd and Crisp[36] stated that in 820,000 admissions to the Mayo Clinic, only twenty-five mesenteric tumors were found and of these only eight were cysts, an incidence of less than one in 100,000 admissions.

2. Roller[40] quoted the following reports: The Massachusetts General Hospital has a record of only six cases from 1900 to 1926. The University of California Hospital has a record of one cyst and one tumor of the mesentery in 93,511 admissions since 1906. The Children's Hospital of Los Angeles has three cases, occurring in the years 1932 to 1935, in a total of 12,425 admissions during the same period. Los Angeles General Hospital has a record of one case in 188,921 admissions in the five-year period, 1930 to 1935.

3. Olesen[41] reported no cases found at Los Angeles Hospital from 1912 to 1929.

4. Costello[17] reported one case in 28,312 admissions to Women's and Children's Hospital of Toledo, Ohio.

TABLE I

DATE	SURGEON	CONTRIBUTION
1507	Benevieni	First observed a mesenteric cyst at autopsy
1803	Portal[22]	Classified these cysts
1842	Rokitansky[38]	First described chylous cysts from autopsy
1880	Tillaux	First successfully operated on a cystic mesentery tumor[1]
1883	Pean	First marsupialized such a tumor successfully and later Bramann, Kilian, Millard, and Marklen also treated patients surgically[1]
1886	Augagneur[23]	Found 18 out of 90 cases of mesenteric tumors to be cystic
1887	Hahn[24]	Classified cysts as blood, chylous, serous, and echinococcus
1891	Arekion[25]	Referred to 81 case reports
	Bianchi[35]	Found 95 cases to this date
1892	Braquehaye[26]	Added 23 cases and said the total to date was 104
1897	Moynihan[27]	Added 9 cases and reviewed the literature
1900	Dowd[9]	In a classical review added 32 published cases making the total 145
1906	Porter	Estimated the total number of reported cases at 200[1]
1910	Timball[11]	Added 30 cases
1912	Friend	Collected 52 cases of chylous cysts[1]
1913	Benedict	Added 44 cases
1921	Carter[19]	Stated that not over 300 cases had been reported
1924	Higgins and Lloyd[14]	Reported that 250 cases had been recorded
1930	Flynn[20]	Judged that there were 200 to 300 cases published
1932	Warfield[1]	Found 129 cases reported since 1920 and estimated total to be about 500
1941	Loeb[3]	Estimated 550 to 600 cases reported

5. Porter[42] stated that no mesenteric cysts were found in 15,000 autopsies at the University of Minnesota and only two cases were observed in 200,000 clinical case reports at St. Mary's and St. Luke's Hospitals in Duluth, Minn.

6. Roller[40] found three cases in 900 surgical admissions to the University of Pennsylvania in two and one-half years (1931 to 1933).

7. As far as we can determine, our case is the third one of a mesenteric cyst in about 750,000 admissions to Mercy Hospital, Chicago, in the past ninety-eight years. Hueper[43] reported a case of mesenteric cyst (mesenteric enterocystoma) from this hospital in 1927. The patient was a boy, aged 5 years, who had a cyst the size of a hen's egg, located between the leaves of the mesentery 2½ inches from the ileocolic juncture, but not connected to the ileum. A third case was reported by Sawyer[44] in a personal communication. This case, however, was never reported in the literature.

TABLE II. FOUR HISTORICAL PERIODS OF MESENTERIC CYSTS ACCORDING TO BRAQUEHAYE[26] AND OTHERS

PERIOD	DATE	CHARACTERISTICS OF PERIODS
1	1507-1850	The lesion was found only at autopsy
2	1850-1880	Occasional operations were performed but usually on an incorrect preoperative diagnosis and none of the patients is reported to have survived
3	1880-1900	Operations for this lesion were followed by a few recoveries
4	1900-Date[34]	The condition was admirably described by Dowd, and surgeons took cognizance of such an entity and diagnosed several cases preoperatively

Therefore, it is reasonable to say that mesenteric cysts are sufficiently uncommon to arouse considerable interest. Warfield[1] emphasized that many surgeons of wide experience have neither observed nor operated upon a patient. The consensus of opinion in the literature is that the condition occurs twice as frequently in women and is rarely found in the colored races. Of all mesenteric tumors the cystic variety is four times more common than the solid. Most textbooks on surgery refer to the subject only briefly, if at all. Surgeons compose the majority of authors who write on this subject and most of them report a single case. Several have reported two cases; Roller[40] had three and DePenna[21] reported four. Hinkel[16] and others stated that the diagnosis is rarely made before surgery but this is not entirely so since diagnosed cases are reported by Haworth,[28] Bertolini,[29] Naumann,[32] Aloi,[30] Finucci,[31] Levison and Wolfsohn,[33] Kross,[15] and others.

ETIOLOGY AND CLASSIFICATION

Various classifications of mesenteric cysts have been offered as aids to a better understanding of the subject. Portal[22] was the first to make such a proposal in 1803. In 1842, Rokitansky[38] said that the tumors arose from degenerated lymph nodes. Braquehaye[26] listed the cysts according to their contents as sanguineous, lymphatic, parasitic, etc. In 1897, Moynihan[27] elaborated somewhat on this same classification and suggested several causes for the formation of these cysts. Dowd[9] in 1900, first attempted to name the various types of cysts according to their origin. It was his opinion that many of the cysts were of embryonic origin and he presented much evidence in proof of this opinion. He further stated that we can include all mesenteric cysts in three categories: (1) embryonic cysts, (a) hydatid cysts, (3) cystic malignant disease. Dowd also urged all investigators to obtain microscopic and chemical analyses of these cysts so that we can better understand the problem.

Many authors since have amplified Dowd's classification and the literature contains many theories as to the genesis of mesenteric cysts but the subject is still rather confusing. For a more detailed version of this phase of the problem we refer the reader to an original article by Warfield,[1] who presented an excellent discussion of the entire subject. More recent ones are Swartley,[10] Loeb,[3] Lahey and Eckerson,[2] Berger and Rothenberg,[13] and Roller,[40] who feel that mesenteric, omental, and retroperitoneal cysts should be classed together. Hill (quoted by Dunne[7] and Larkin[12]), in 1930, presented a classification which is widely accepted at present. This is also a modification of Dowd's classification. Hill said that all mesenteric cysts are of two types:

A. Simple
   1. Serous
   2. Chylous
   3. Irregular types

B. Neoplastic
   1. From ectoderm (dermoid)
   2. From mesoderm (lymphangioma)
   3. From entoderm (enterocystoma)
   4. From fetal inclusions (teratoma)

Newer work on these cysts reveals that some are due to diverticula from the intestine in the embryo. It was suggested by Guthrie and Wakefield[4] that

these diverticula may extend into the mesentery and become pinched off. Some are absorbed and others remain to form cysts. Intestinal epithelium was found in the majority of twenty-two human cysts and according to these authors substantiates the theory of embryonic origin of mesenteric cysts.

SIGNS AND SYMPTOMS

Mesenteric cysts may occur anywhere from the duodenum to the rectum. Over one-half of the cysts occur in the small bowel and one-fourth of them in the mesentery of the ileum.[1] The contents of the cysts may be clear, milky, sebaceous, bloody, yellow, or brown. These cysts are usually benign but malignant degeneration can occur and may be either carcinoma or sarcoma. Recently, there have been several reports describing such changes.[6, 37]

Fig. 1.—Scout roentgenogram of abdomen showing calcified mesenteric cyst in left upper quadrant.

Other than pain the symptoms are those due to complications, principally obstruction. Roller[40] stated that acute, chronic, or intermittent intestinal obstruction is responsible for 40 per cent of all complications. Pain is present more frequently in this type of cyst than in any other type of cystic abdominal tumor. Increase or decrease of the patient's weight is not of any great significance. An abdomen in acute surgical condition may present itself if rupture of a mesenteric cyst occurs. A careful history will reveal the probable cause of such an episode.

DIAGNOSIS

The principal point in the diagnosis of mesenteric cysts is to bear in mind the possibility of such a condition causing the symptoms present in a patient

Fig. 2.—Roentgenogram of barium enema showing mass outside gastrointestinal tract.

Fig. 3.—Roentgenogram following evacuation of enema.

Fig. 4.—Intravenous pyelogram showing mass outside the kidney—ureter—bladder tract.

Fig. 5.—Lateral roentgenogram further localizing the calcified mass.

with an abdominal mass. Many grow to a fairly large size with the patient complaining only of an increase in girth of the abdomen. A rounded abdominal mass, which may be soft or firm, tender or not should at least suggest the possibility of a mesenteric cyst. A single scout film of the abdomen is seldom of great assistance in the diagnosis since many of these cysts are not radiopaque and cast no shadow. Very occasionally, as in our case, the cyst wall contains sufficient calcium to be outlined on the x-ray plate (Fig. 1). One of the cysts in Hinkel's report[16] also contained calcium. It is important to rule out a connection with the bowel lumen, which can be done with a barium meal or enema (Figs. 2 and 3). A pyelogram will aid in ruling out any connection with the kidney (Fig. 4). Both anteroposterior and lateral films aid in locating the cyst (Fig. 5). Hinkel also suggested pneumoperitoneum as a possible diagnostic aid. These results plus a careful history and physical examination should lead to a correct diagnosis in most instances.

TREATMENT AND MORTALITY

Treatment appears to be the one phase of the subject that most authors agree upon. In the order of desirability the treatment is as follows:

1. Total removal or enucleation is the operation of choice.

2. Enucleation with subsequent intestinal resection made mandatory by involvement of the blood supply to a portion of the bowel.

3. Marsupialization is the least desirable method and is resorted to only when point 1 or 2 is not possible due to too extensive involvement of the bowel. A draining sinus often accompanies this procedure but usually heals in one to three months.

4. Aspiration is mentioned only to be condemned as a method of treatment. It may be used for diagnostic purposes after the tumor has been isolated at operation, or to facilitate removal by reducing the size of the cyst.

Atchley[39] reported the mortality as a whole to be about 35 per cent. Roller[40] stated that the mortality following removal of the cyst without intestinal resection is about 9 per cent; with resection 25 to 30 per cent; and with marsupialization about 16 per cent.

CASE REPORT

E. M., aged 32 years, a white woman, para i, gravida i, entered Mercy Hospital, Jan. 20, 1946. The patient was well up to four to five years prior to hospital admission. At that time she complained of headaches associated with vomiting. Medical work-up resulted in the conclusion that no migraine was present. This cleared up gradually and three years before admission she began to have pain in the region of the sixth thoracic vertebra which radiated superiorly to the occiput. This pain was not related to the previously mentioned headache. A physician consulted at this time suspected some focus of infection but the patient refused another work-up and became well in six to eight weeks without medication. Seven years before admission the patient was in an automobile accident and sustained contusions and abrasions to the left leg, thigh, hip, and shoulder and left side of the chest and a fractured nose. Six months before she noticed a "shooting pain" which began in the left sacroiliac region and left lower quadrant of the abdomen and radiated down the anterior aspect of the thigh to the knee.

The past history was negative except as described. The family history was noncontributory.

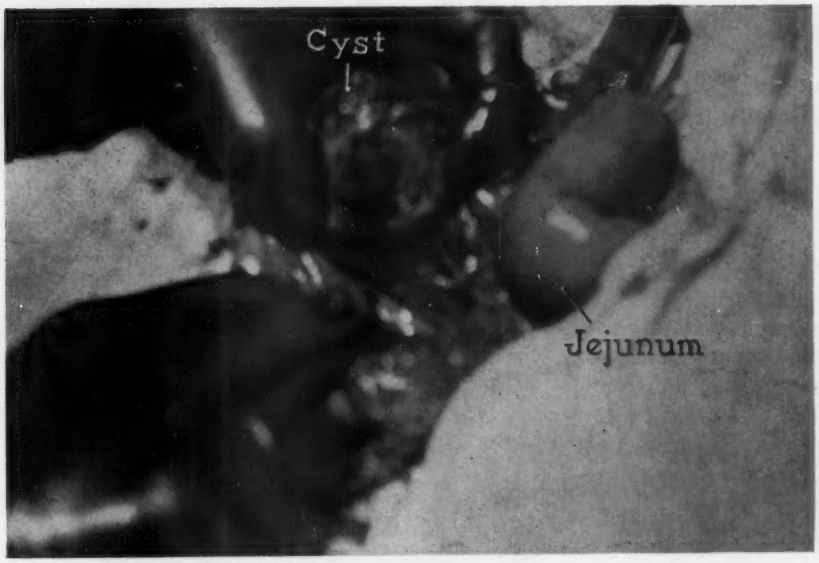

Fig. 6.—Photograph at operation showing calcified mesenteric cyst being dissected from its bed near the duodenojejunal ligament (Treitz).

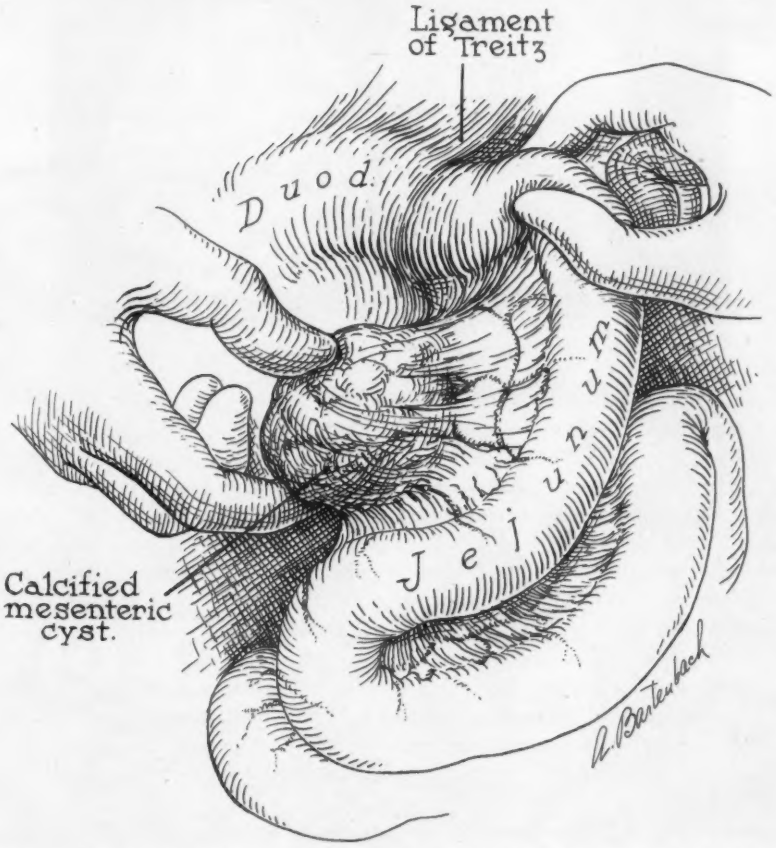

Fig. 7.—Artist's reproduction of photograph, after the mass was separated from the jejunum, showing relation of calcified mesenteric cyst to duodenum, jejunum, and ligament of Treitz.

The essential findings on physical examination were in the abdomen, which was soft, with tenderness elicited in the left upper quadrant. There was no rigidity except voluntary when the left upper quadrant was palpated. There was a firm, smooth, freely movable mass about the size of an orange just to the left and above the umbilicus. All tenderness was centered about this mass. The mass was more easily palpable when the patient lay on the stomach, whereupon the mass moved forward and lay against the anterior abdominal wall. The liver, kidneys, and spleen were not palpable.

Because of the firmness it was thought that the mass was calcified and the impression was listed as calcified cyst or hematoma of mesentery. A pedunculated pancreatic cyst, hypernephroma of left kidney, calcified accessory spleen, and retroperitoneal sarcoma were to be ruled out.

Urologic Consultation.—Pyelogram revealed a calcified cyst not attached to the kidney. The impression was calcified mesenteric cyst.

Laboratory Reports.—There were red blood cells, 4,380,000; white blood cells, 6,000; hemoglobin, 13 Gm. (84.4 per cent). Urine (voided specimen) was negative except for six to ten white blood cells per high-power field and rare red blood cells. Blood chemistry was essentially normal with nonprotein nitrogen 28.5, urea nitrogen 14, creatinine 1.6, chlorides 480, sugar 73, calcium 10, serum protein 6. Basal metabolic rate was minus 21 per cent.

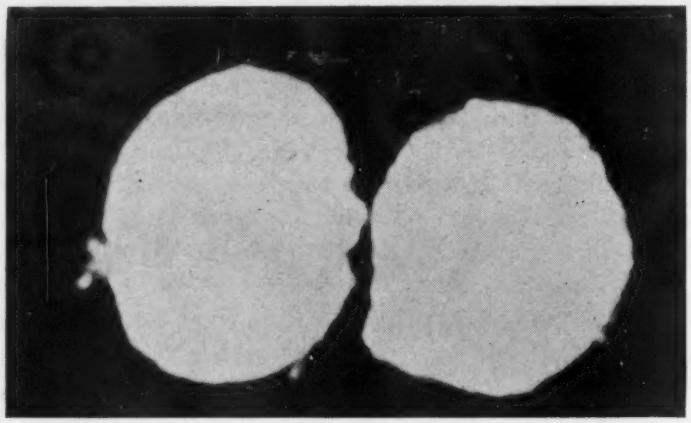

Fig. 8.—Photograph of calcified mesenteric cyst.

On Jan. 25, 1946, exploration of the abdomen was carried out. A left paramedian incision was made and the tumor was found in the left upper quadrant, surrounded by omentum and adhesions. Careful dissection revealed the mass to arise from the base of the jejunal mesentery close to the ligament of Treitz. Although the jejunum was adherent to the superior aspect of the mass, further dissection revealed it to be an inflammatory attachment and not a true outpouching of the jejunal wall (Figs. 6 and 7). Removal of the cyst was carried out uneventfully and no opening into the bowel could be demonstrated. The serosa of the jejunum was closed with interrupted silk sutures and hemostasis was secured without involving any of the major vessels to the bowel. The appendix was quickly removed and the abdomen closed in layers without drainage.

The patient had an uneventful convalescence except for a mild cystitis due to a postoperative catheterization which quickly cleared up. She was ambulatory on the eighth postoperative day and left the hospital on the twelfth postoperative day. Follow-up twelve months postoperatively revealed the patient working every day, although still complaining of occasional headaches associated with nausea.

Pathology.—The cyst was semisolid in consistency and roughly ovoid in contour, measuring 5.2 by 4.4 by 3.8 cm. (Fig. 8). The outer aspect of the cyst was smooth in areas and elsewhere shaggy reddish-gray. On sectioning, a fairly thick wall measuring up to 0.3

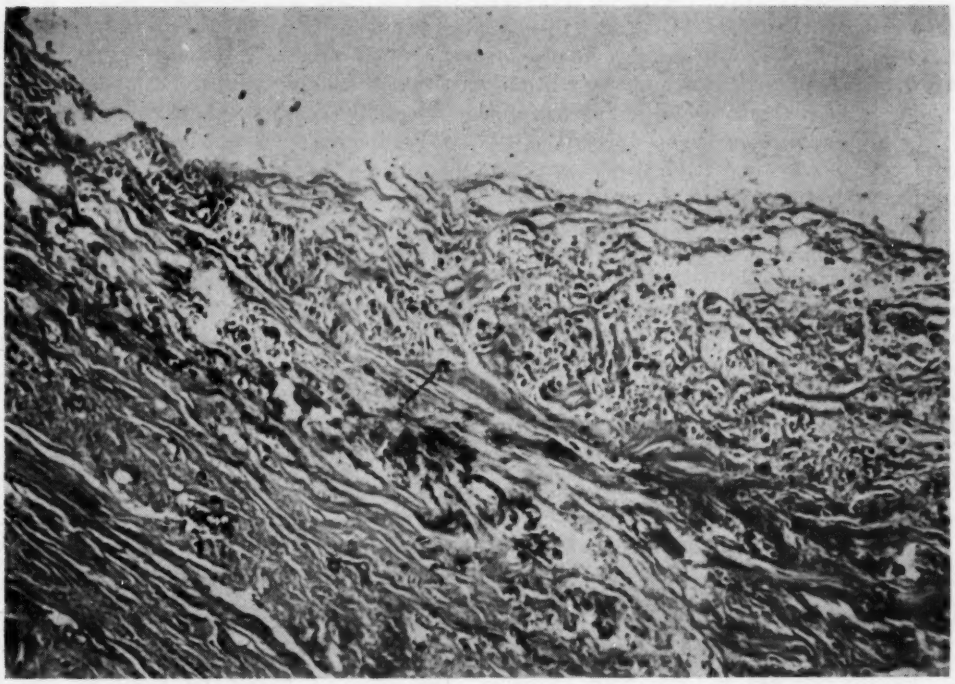

Fig. 9.—Section through inner half of cyst wall (×260): Prominent calcium deposition; dense collagenous fibrous tissue wall, and "frayed" inner aspect with chronic inflammatory and fat-laden macrophages within interstices of connective tissue.

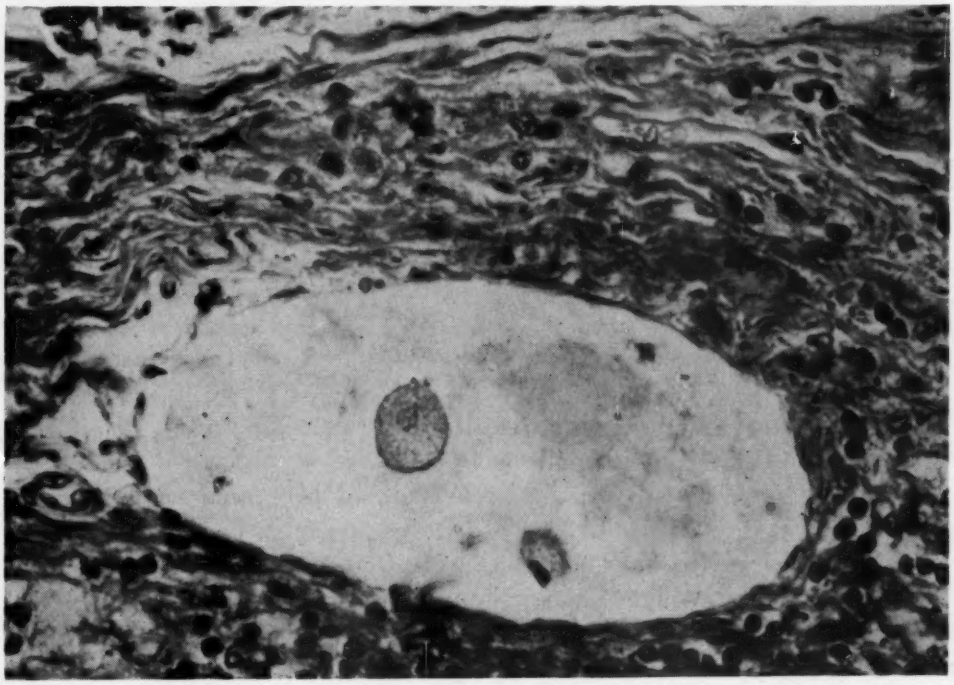

Fig. 10.—Section through endothelial lined lymphatic channel (×675) with two fat-laden "foamy" macrophages; chronic inflammatory cells in dense collagenous tissue of cyst wall is shown.

cm. was revealed. In many areas the wall was quite rigid and sectioned with a distinct grating sensation due to gritty calcific deposits; elsewhere the wall was densely fibrous in nature and pliable. The interior of the cyst was filled with soft, yellowish-gray material of the consistency of cottage cheese and slightly greasy. Removal of the contents disclosed a fairly smooth, grayish-white to pale tan inner lining with occasional roughened areas.

Multiple microscopic preparations of the cyst wall revealed it to be composed of fairly dense collagenous connective tissue with scattered foci of chronic inflammatory cells, predominantly lymphocytes, with occasional plasma cells, monocytes, and neutrophiles (Fig. 9). Calcific deposition occurred irregularly throughout the innner half of the wall. Fat laden, "foamy" macrophages were prominent in many areas along the innermost aspect of the wall and within a few endothelial lined channels (Fig. 10). The latter were situated at the outer confines of the wall. Formidable lymphoid tissue aggregates were nowhere apparent and the inner expanse of the wall was devoid of identifiable endothelial or epithelial lining elements. In the smoother portions of the interior aspect, condensations of collagenous fibrous tissue were apparent but no particular affinity for silver stains was demonstrable. Smears and cultures of the cyst content in addition to Gram-Weigert and modified Ziehl-Neelsen preparations of various aspects of the wall disclosed no organisms.

COMMENT

It is our impression that this cyst was of the lymphogenous type because of the inspissated chyliform content and prominent lymph channels in the wall of the cyst, some with "foamy" fat-ingested macrophages.

Due to the appearance of the contents of the cyst an underlying tuberculous factor was considered; however, the lack of caseative features to the contents, the failure to demonstrate tubercle bacilli, and the lack of tuberculous granulation tissue in the wall tend to rule out tuberculosis.

If this cyst were of the enterogenous type we would expect to find epithelial inclusion from the intestine plus a more mucoid type of content.

Because of the history of trauma several years previously the possibility of traumatic fat necrosis of the mesenteric fat and pseudo-encystment or localized hematoma were considered. The lack of altered blood pigments and foreign body giant-cell reaction in the cyst wall mitigate against these possibilities.

SUMMARY AND CONCLUSIONS

1. A case of mesenteric cyst is reported as the first calcified mesenteric cyst in ninety-eight years at Mercy Hospital, Chicago, in over 750,000 admissions.

2. Mesenteric cysts are rare and calcified mesenteric cysts are extremely rare.

3. The history of these cysts dates to 1507, when they were first found at autopsy and considered "anatomic curiosities."

4. Their history is divided into four periods, beginning with 1507 to the present.

5. The etiology, pathology, and classification remains obscure.

6. Cysts in the region of the jejunum and upper ileum are chiefly mesenteric in distribution, while in the ileocecal region they lie in the submucous or muscular layers of the intestinal wall.

7. Complications are serious. An acute abdomen may be the existing condition when first seen by the surgeons.

8. Mesenteric cysts must be included in differential diagnosis of obscure abdominal pathology.

9. It is possible to diagnose these cases if a careful history, physical examination, and laboratory checkup are done.

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Book Reviews

Handbook of Fractures. By Duncan Eve, Jr., M.D. Pp. 263, with illustrations. St. Louis, 1947, The C. V. Mosby Company.

This handbook was prepared by the author for students, practitioners, and surgeons in general, omitting lengthy discussions of the manner of injury together with the signs and symptoms of fractures, and placing emphasis on the details of treatment. The text is adequately illustrated with photographs, roentgenograms, and drawings.

The author devotes three chapters at the beginning to immobilization methods, open reduction and internal fixation, and compound fractures; the remainder of the handbook is then broken up into chapters dealing with fractures in the various regions of the body. In general, the author outlines the one or two procedures which he himself generally uses in connection with a given fracture, and therefore has found from experience to be satisfactory. In those instances in which open reduction seems indicated, he outlines briefly the technique and procedure for the operation.

The book is interestingly written and well illustrated; the methods and procedures described are basically sound and conservative. As one reads and reviews it, he senses that the author is writing from a wealth of experience and that his judgment is mature and sound. It will prove a useful guide to anyone undertaking to deal with the average run of fracture problems in the hospital or in civil practice.

Outline of the Spinal Nerves. By John Favill, Clinical Professor of Neurology, University of Illinois. Pp. 190, with 12 illustrations. Springfield, Ill., 1946, Charles C Thomas, Publisher.

This book is composed for the most part of lists of nerves and muscles with certain information about each.

Part I consists of a list of the spinal nerves with the muscles innervated by the dorsal and the ventral division of each.

Part II is composed of lists of all nerves with the muscles supplied by each nerve, the segments contributing to the innervation of each muscle and other information about lesions of each nerve.

Part III lists all muscles with the nerves which innervate them, also the segmental innervation of each muscle, also its origin, insertion, action, function, test for loss of function, and the effect if it is paralyzed.

Part V is thirty-three pages of what the author calls supplementary data.

It is obvious that this is a reference book. Most of the material in it is anatomic detail learned by the freshman medical student in Gross Anatomy but soon forgotten, reviewed by the neurologist in training, but again only partially retained. It should be a great convenience to the neurologist or neurosurgeon to have these data collected and condensed in this form for easy reference.

Five Million Patients. By Allen W. Freeman, M.D. Pp. 299. no illustrations. New York, 1946, Charles Scribner's Sons. $3.

This is the autobiography of the author beginning with his entrance into medical school in 1901 and ending presumably with his retirement from Professor of Public Health Administration at Johns Hopkins in 1945.

After graduation in 1905, and one year of internship, he engaged in private practice until June, 1907, when he was appointed City Medical Inspector of Richmond, Va. He remained in public health work, becoming Assistant Commissioner of Health for the state of Virginia in 1908, Epidermiologist in the United States Public Health Department in 1915, Commissioner of Health of Ohio in 1917; he was a major in the Reserve Corps of the Army from July, 1918, to December, 1918, and then resumed his duties as Commissioner of Health of Ohio until he was appointed Resident Lecturer in Public Health Administration at Johns Hopkins in 1921. After one year as lecturer he became Professor at Johns Hopkins, where he remained until retirement in 1945.

The book consists of 111 chapters, each relating some episode in the author's life which he considered important or at least interesting. Physicians will find it more entertaining than enlightening, but it does give an account, easy to read, of many phases of public health in an earlier period.

Those untrained in medicine and also public health nurses, social workers, etc., will find this book interesting and profitable reading.

COMMUNICATION

THE NEW YORK HOSPITAL
JANUARY 8, 1948.

ALTON OCHSNER, EDITOR
OCHSNER CLINIC,
NEW ORLEANS, LA.

DEAR DR. OCHSNER:

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